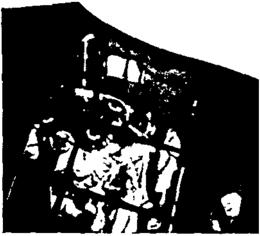
Fundamentals of General Surgery



Fundamentals

OF

General Surgery

JOHN ARMES GIUS, M.D., D.Sc. (Med.), F.A.C.S.

Professor of Surgery, College of Medicine,
State University of Iowa

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Preface

AS THE BOUNDARIES of medicine steadily expand, the problems of medical education become more complex. The concept that education is a dynamic endeavor which must be continuously adapted to changing times is applicable to the over-all problem of medical progress. However, the undergraduate period in medicine is short and the fund of medical knowledge which it appears desirable to bring to the student is voluminous. To be realistic, it is impossible to encompass the field of medicine in its entirety in the undergraduate years. Therefore it is time to clarify objectives, to select and organize relevant material and to determine how these data may best be presented to the student. In order to do this, we must determine what the undergraduate should learn and what the graduate may reasonably be expected to learn after leaving medical school. It is apparent that the education of the physician is a continuing process and that graduation from medical school is only one milestone in this process. We cannot expect the medical graduate to be a finished product from the mere circumstances of his exposure to a vast amount of accumulated knowledge, some laboratory and clinical experiences and a sincere effort to understand the human body and its ills. While some of the knowledge offered at medical school is retained, it is soon forgotten unless used. It seems, therefore, that we must determine and teach that which we consider *basic* and convey with it an understanding of the implications, correlations and applications to medicine in its many phases, now and in the foreseeable future. We can hope to accomplish little more than this at the undergraduate level.

In the present work, the attempt has been made to set forth in simple terms the considerations that are believed to be fundamental to the study of general surgery, including certain aspects of physiology and pathology encountered in the patient. Detailed descriptions of

technical procedures (e.g., operations) which serve to confuse, rather than to clarify, the student's thinking have been omitted. Such items are best presented in the postgraduate years. Pathophysiologic mechanisms as they apply to an understanding of the surgical patient have been emphasized, and the holistic concept in the care of the patient and the dangers inherent in the narrow and uncritical perspective of the all too common surgical technician have been stressed.

The author believes that undergraduate teaching in medicine in general and in surgery in particular need not suffer serious embarrassment from the limitations of time allowed by present-day curricula. Given a student who has received proper motivation and has been exposed to the basic knowledge of the premedical and medical curriculum, there need be no fear that items left unsaid will necessarily remain unlearned

The textbook serves a limited function in the teaching-learning processes in medicine. It is inevitable that some deficiencies attach to every effort to represent a dynamic process in static form, of which textbooks are examples. Furthermore, the ideas expressed in any work, such as this one, reflect only the concepts and experiences of the author The author's ideas also change, and not infrequently they are at variance with the ideas of others. Thus the present work is only an attempt to describe within the limitations of time, space, knowledge and language those concepts which the author and his contributors believe and teach. It is not pretended that these discussions are complete, on the contrary, it is recognized that they are quite incomplete. For this reason, the student must also be exposed to the problems of the patient in the hospital wards and clinic, to ward rounds, lectures, conferences and personal discussions with his instructors and fellow students. Above all, he must himself be an active participant. Each such experience tends to augment other experiences.

The objectives of undergraduate surgical teaching listed below have evolved from many classroom and bedside efforts and many discussions with colleagues and students It is hoped that this book may aid the student in their attainment.

1. To advance growth and understanding regarding fundamental preclinical and clinical knowledge and to increase the student's capacity for the application of basic principles.
2. To increase his awareness of the great variety of clinical forms produced by a given pathologic process.

3. To develop sureness and equanimity in the conduct of emergency situations.
4. To become acquainted with various alternative managements of given problems.
5. To increase knowledge and confidence in handling certain "minor" surgical problems.
6. To gain a "surgical approach" to the patient by developing specific management technics applicable to the preoperative, operative and postoperative periods.
7. To learn what may be predicted of certain surgical procedures, in terms of hospital care, cost and time, "cure" potential, future complications, disability or recurrence. Long-range follow-up in the clinic for outpatients is especially important in this regard.
8. To augment the development of satisfactory doctor-patient and doctor-doctor relationships.

—John Armes Gius

ACKNOWLEDGMENT

I am grateful to many of my associates for the encouragement and assistance they have given me. I am especially indebted to Dr. Clare G. Peterson and the late Dr. Thomas M. Joyce of the University of Oregon Medical School, where this work was begun, as well as to Dr. Robert T. Tidrick, Head of the Department of Surgery of the State University of Iowa, for innumerable valuable suggestions.

Certain sections have, in large measure, been written by others. These sections include: Fluid and Electrolyte Balance, Dr. Edward E. Mason; Fractures, Dr. Edgar S. Brintnall; Care of the Patient in the Operating Room, Dr. Stuart C. Cullen; and Head Injuries, Dr. Russell Meyers. To these men I give special thanks.

In addition, many have contributed material or have reviewed the manuscript and proposed important changes. These contributors include: the late Dr. Kate Daum (Nutrition), Dr. Sidney E. Ziffren (Burns), Dr. Robert C. Hickey (Tumors), Dr. James A. Clifton (The Liver), Dr. Henry E. Hamilton (The Spleen), Drs. Elmer L. DeGowin

and Joseph A Buckwalter (The Thyroid), Dr. William C. Huffman (The Head and Neck), Dr. Rodman E. Taber (Peripheral Vascular Diseases, Chest Injuries) and Dr. Albert P. McKee (Infections), all of whom are on the staff of the State University of Iowa, and Dr. Cushman Haagensen of Columbia University (The Breast). I am deeply indebted to all.

I have also had the invaluable assistance of Mr. Paul Ver Vais and his staff of the Department of Medical Illustration, and the co-operation of Mr Fred Kent and his staff of the Photo Service in procurement of the photographs. Many of the radiographs have been graciously supplied by Dr. Carl L. Gillies of the Department of Radiology To all these I give sincere thanks.

I must also acknowledge my gratitude to the secretaries who have typed and retyped the manuscript, which never seemed to be in final form. They have given of their efforts unstintingly and deserve much more credit than I can express.

During the years that "the book" has been in the making, my wife, Martha, has been a constant source of inspiration and assistance, without which the task would never have been completed.

Finally, The Year Book Publishers, and especially Mr. Fred A. Rogers of that organization, have been most patient, helpful and loyal. To the publishers I am grateful.

—John Armes Gius

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CHAPTER 1

Historical Perspectives

THE HISTORY of surgery can be traced to ancient times; but from these early beginnings, through the Dark Ages, the Middle Ages and the Renaissance, little progress was made in surgery as we know it today. For some thousands of years, it was a combination of mysticism and religious rite. Skulls were opened for the release of demons; circumcision was a priestly rite. The first of the specialists was called into being to cut for the stone. Always, in this world of violence, there was the surgery of trauma. And paralleling man's inventiveness were the increasingly destructive exigencies of war. The surgeon was a craftsman of a sort, meeting such problems with the red-hot rod, boiling oil, the septic ligature or amputation. Surgery was a bitter task, abused by charlatans, outlawed at times by the body politic of organized religion, with suffering and death its consequences.

Medicine as a science dates to the Renaissance and the exciting discovery that the experimental method was the key to new knowledge. Search was the spirit of the age. Men were possessed of the idea.

> *Let thy Studies be free as thy Thoughts and Contemplations; but fly not only upon the wings of Imagination; Joyn Sense unto Reason, and Experiment unto Speculation, and so give life unto Embryon Truths, and Verites yet in their Chaos*
>
> —SIR THOMAS BROWNE, 1605-1682
> From Essay on "Christian Morals,"
> first published 1716.

Copernicus, Da Vinci, Galileo, Napier, Leeuwenhoek, Bacon, Vesalius and Harvey are numbered among its standard-bearers. The development of the microscope and the telescope, the broad implications of the law of falling bodies and the science of logarithms provided man

Ambroise Paré (1510-1590), French army surgeon, chronicled his war experiences and, with inquiring mind, raised again and again the inevitable "Why?" Then, in wisdom and humanity, based on his own observations and experience, Paré rejected the cautery and boiling oil for more beneficent methods of treatment: *Je l'ai pansay: Dieu le gauril!* *

Fundamental advances in science and medicine continued in the centuries that followed. The nineteenth century is notable for the evolution of concepts and the development of technics that are the basis of modern surgery. Virchow (1821–1902) established the cellular basis of pathology. Louis Pasteur demonstrated that fermentation is dependent on living organisms (1861). Koch elaborated his famous postulates regarding the bacteriologic specificity of disease: "One microbe, one disease." It seemed clear now that infections did not arise *de novo* and without cause. The setting was complete in anticipation of Joseph Lister's memorable contribution to mankind; *On the Antiseptic Principle in the Practice of Surgery* was published in 1867. The concept of homeostasis, the outstanding generalization of biology, was presented to medicine by the French physiologist Claude Bernard (1813–1878), whose brilliant summarization of this remarkable idea is quotable in one short sentence: *La fixité du milieu intérieur est la condition de la vie libre.*† The basic philosophy and strict requirements of the scientific method were given classical statement by this same Claude Bernard in *La Science Expérimentale* (1878). Toward the close of the century, Röntgen discovered the x-ray and the Curies isolated elemental radium.

When all of this, and more, had been realized, the scientific bases of modern surgery were secure. And what of operative surgery through all the long centuries?

The status of operative surgery was clouded in darkness. Scarcely longer ago than one man's lifetime, surgery was shock and pain and sepsis. In the middle of the nineteenth century, wounds rotted and stank. The surgeon Pirogoff (1854) gave expression to his own agonizing sense of futility, in an essay on luck in surgery, estimating his skill and methods of treatment as nothing compared with that of chance in determining the success of an operation. Wound suppuration, purulent edema, erysipelas, traumatic tetanus and hospital gan-

* I dressed the wound, God healed it!

† The stability of the internal environment [the body fluids] is the condition of the free [independent] life.

with the tools of discovery and revealed new worlds to conquer. The development of the printing press—mortal foe of dogma—furnished the means of communication whereby knowledge and discovery might become the common property of all men.

In 1543, Vesalius, Professor of Surgery at Padua, effected a one-man revolution in medicine with the publication of *De Corporis Humani Fabrica Libri Septem.* Science was brought to life in the study of anatomy. Benievieni, Malpighi and Valsalva soon demon-

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JOSEPH LISTER
1827–1912

strated that the postmortem examination could reveal the hidden causes of disease, and the science of clinical pathology was born. William Harvey introduced the experimental method into medicine in 1628 with the publication of *De Motu Cordis et Sanguinis in Animalibus* and John Hunter (1728–1793) extended it to the study of surgery.

Men of medicine spoke for their age. In symbolic act, Paracelsus (1493–1541) burned the books of Galen and Avicenna and then instructed his students to study, to observe and to experiment.

in 1886, and instruments could be freed of bacteria. The operator's hands could be scrubbed, and the patient's skin could be cleansed with soap and water. By 1894 the rubber glove of Halsted was used to exclude the hands of the surgical team from the operative wound. Johann von Mikulicz-Radecki studied the bacteriology of air and suggested the use of surgical masks. As the aseptic technic developed, the need for antiseptics decreased. Soon the reorientation was com-

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WILLIAM STEWART HALSTED
1852–1922

plete. In 1892 the fixed and timeless principles of asepsis were summarized in one small volume by Curt Schimmelbusch, entitled *The Aseptic Treatment of Wounds.*

The *control of hemorrhage* was a pyrrhic victory for surgeons until anesthesia and asepsis made deliberate surgery and wound healing by first intention a probability rather than an accidental occasion. The art of hemostasis was slowly learned. Ambroise Paré, four hundred years ago, discarded the red-hot rod, boiling oil and

grene were the scourges of surgeons. Little more than a dozen years before, anesthetics were unknown and surgery was pain. Little wonder that a most extreme emergency was required for a patient to submit to the knife. Then, almost in an instant, operative surgery was transformed by three remarkable developments: the introduction of anesthesia, antisepsis and asepsis, and the control of hemorrhage.

The *story of anesthesia* is a fascinating chapter in the history of medicine. Modern anesthesia began with Crawford Long of Jefferson, Georgia, who, from observations at an ether frolic, was stimulated to give ether by inhalation to one James Venable for the removal of a tumor on his neck (1842). This is a memorable date in history, although the details of Long's studies were not published until 1849. In the meantime, a historic demonstration had taken place: On October 16, 1846, in the surgical amphitheater of the old Massachusetts General Hospital, William T. G. Morton, dentist and medical student, administered an unrevealed vapor (ether) to Gilbert Abbott for the removal of a tumor on his jaw (surgeon, John Collins Warren). Subsequently, the term "anesthesia" was coined by poet-physician Oliver Wendell Holmes. The value of nitrous oxide for anesthetic purposes had been shown in 1844 by Horace Wells of Hartford Chloroform was given strong public support after Sir James Young Simpson, Professor of Midwifery at the University of Edinburgh, had given it to Queen Victoria when Prince Leopold was born (1853). Some dozen years after the discovery of ether the conquest of pain was *fait accompli.*

Antisepsis and *asepsis* were anticipated by the epochal studies of Louis Pasteur and Robert Koch during the years 1850–1865. The combined results of their efforts were to shatter the ancient theory of the spontaneous generation of infections and to pave the way for Lister Lister's experiments and clinical trials with the application of strong carbolic solutions to freshly received compound fractures were reported in 1867. Carbolized dressings were introduced, and carbolic spray for use in operating rooms. The antiseptics purified the surgical field but were necrotizing to patient and surgeon alike. To protect the hands of his surgical nurse, William Stewart Halsted introduced the use of rubber gloves in surgeries. The obvious disadvantages of carbolic acid led to an extension of the Antiseptic Principle. If purification of the wound could be achieved by antisepsis, protection of the wound might be obtained by preventive measures. Sterilization by boiling was introduced by von Bergmann

tourniquet, the electrocautery and the silver clip, as the surgeon strives to achieve the ideal state of complete hemostasis.

MODERN SURGERY

As one looks backward, the brightness of the nineteenth-century revolution and the dates 1842 and 1867 obscure all lesser milestones. The so-called *surgical phase* of modern surgery was in large part the special contribution of the fifty years following these advances.

The first result of anesthesia and asepsis was a reduction in the mortality rate of operations that had been done since time immemorial: amputations and vascular ligations, excision of superficial tumors, emergency operations for strangulated hernia, breast amputation, removal of bladder stones, trephining, etc. The surgical technics of the past were modified and perfected. The scope of surgery was then extended to new frontiers. Elective surgery became a practical reality, and visceral surgery was born. Open procedures on bone and joints, reconstructive operations of all types, surgical procedures on the central nervous system, thoracic surgery and, more recently, the spectacular advances in cardiovascular surgery, carried operative surgery to the last of the anatomic frontiers by the middle of the present century (1950). Modern surgery with the ultrasonic scalpel, for example, first used to approach subcortical and midbrain areas of the central nervous system in 1955, is a far cry from cold steel. And modern closed-circle-system anesthesia, with induced hypothermia and various cross-circulation, oxygenating heart-lung devices, to allow direct access to the inside of the heart, are a world away from the occasion of using an "unrevealed vapor" (ether) for the removal of a tumor on the jaw in 1846.

The true story of any age is to be found in the narrative account of its people—in the personal story of their lives, the record of their accomplishments, their triumphs and failures, their advances and retreats, their hopes and fears. The story of surgery is not to be had for the telling. It is to be found in dusty archives of surgical and medical history, seldom-read biographic and autobiographic accounts of the personalities of the day, a firsthand acquaintance with old leather-bound textbooks and lectures on surgery, handsomely engraved and illustrated with a profusion of woodcuts. Go to these if you would have the history of surgery come alive: To the *Collected Surgical Papers* of William Stewart Halsted, first Professor of Surgery at the

styptics and recommended the use of the ligature—a method of hemostasis at least as old as Avicenna. Marel introduced the principle of the tourniquet into military medicine at the Siege of Besançon (1674). Lister described chromatization of catgut suture material (1876) and produced ligatures that were little different from those of today. The crude antecedents of the modern hemostat fade into the past; but Kocher, Péan and Spencer Wells were using arterial forceps in the latter half of the nineteenth century and devised hemostats, still in use in modern surgeries, which survive under their names. William Stewart Halsted borrowed and refined the surgical craftsmanship of the best Continental surgeons and developed a technology which used fine hemostats and fine silk ligatures. He had this to say about the contributions of the hemostat to modern surgery:

> [Hemostats] determine methods and effect results impossible without them. They tranquilize the operator. In a wound that is perfectly dry, and in tissues never permitted to become even stained with blood, the operator, unperturbed, may work for hours without fatigue. The confidence gradually acquired from masterfulness in controlling hemorrhage gives to the surgeon the calm which is so essential for clear thinking and orderly procedure at the operating table.

The essentials of Halstedian surgery, based on a fundamental knowledge of wound healing, are known to modern-day surgeons as the "Tenets of Halsted"; these are: (1) the gentle handling of tissues; (2) the aseptic technic; (3) sharp anatomic dissection of tissues; (4) careful hemostasis, using fine, nonirritating suture material in minimal amounts, (5) the obliteration of dead space in the wound; (6) avoidance of tension; and (7) the importance of rest. These tenets are the basis of modern surgical craftsmanship.

Two notable contributions to hemostasis in neurosurgery were made by Harvey Cushing in the use of silver clips (1911) and the perfection of the Bovie electrosurgical unit (1928).

The biochemical age has contributed its share, in most notable fashion, to the control of hemorrhage. Chemical fractionation of the components of plasma has led to the introduction of several local hemostatic substances. The products of biochemical research are now available to the surgeon. Purified fibrinogen (Fraction I) and thrombin, combined in the form of foams or films, have proved of value, especially in neurosurgery. Demonstration of the prothrombin-vitamin K relationship has important implications and practical applications in every field of surgery. The biochemical hemostasis of blood clotting has achieved equal importance with the ligature and hemostat, the

hesitant progress in biliary tract surgery until Graham and Cole introduced cholecystography (1921), establishing a rational basis for this type of surgery. To Frederick Treves and the problems of intestinal obstruction. To the exciting monographs on respiration, blood pressure, shock, anoci-association and thyroid disease, filled with protocols, case histories and question marks, of that prescient genius George Crile of Cleveland. To the success story of two young Minnesota surgeons, Will and Charles Mayo, who seized opportunity and founded a surgical empire on the foundation of clinic group practice. To biographic sketches and surgical writings of Bland-Sutton, cockney Napoleon of nineteenth-century London. To the moving and eloquent prose of Berkeley Moynihan of Leeds, gifted and dedicated surgeon, whose defections are lost in the antiquity of time but whose true glory lives on in the form of collected addresses and essays on surgical subjects. To specialization in surgery: Harvey Cushing, Walter Dandy, C. H. Frazier, and Sir Victor Horsley in neurosurgery; Howard Kelly in gynecology, Hugh Young in urology, Robert Jones and Arbuthnot Lane in orthopedics. And to literally scores of others. To fragments of the story in a thousand places. It is, indeed, quite impossible to state all of the facts or to name all of the names.

Surgeon-historians of the present have tried to identify certain characteristics of twentieth-century surgery, so as to distinguish it from that of the nineteenth century. And, in so doing they have indicted the past. During the "surgical phase" of the late nineteenth century, if at times surgery seemed to lose itself and to advance ahead of the basic knowledge required to guide and control it, this relative defection was of understandable origin: the basic information was not yet to be had from the biologic sciences in many an instance, and the new surgical procedures seemed full of hope and promise for the insistent problems of the sick It would seem unconscionably cold and biased to indict nineteenth-century surgery by generalization and in the frame of reference of another century.

It has been the unique and proud contribution of surgeons in every age to recognize the essential unity of medicine and surgery. Throughout the history of medicine, "a most physicianly surgeon" has stepped forward as spokesman for his generation to state the basic truths—that the patient, body and soul, is one and indivisible and that, in the last analysis, the patient must heal himself. The good surgeon of the nineteenth century (as in all other centuries) did not lose his understanding of the patient as a whole in what has been termed a

revivified Johns Hopkins University School of Medicine, for original reference and insight into the personality of the man who epitomized the *Zeitgeist* of his time and was influential in the beginnings of the university tradition in surgery in America. To Kocher of Berne (illustrious predecessor of Halsted), who influenced world surgery by his description of the borrowings and refinements of the basic technics, available to the English-speaking world in the famous translation of his opus on operative surgery by Sir Harold Stiles. To the life and

THEODOR BILLROTH
1829–1894

teachings of Theodor Billroth, father of visceral surgery and cancer surgery, for the statement of the en bloc principle, fundamental premise of all cancer operations, and to the story of the evolution of the follow-up clinic, which, Billroth was aware, is the essential basis of critical evaluation of the results of treatment. To the exciting story of a joining of forces against the ill-defined "typhlitis" by Reginald Fitz, Charles McBurney, and A. J. Ochsner, who accomplished the initial victory over appendicitis. To the accomplishment of Halsted and Bassini, pioneers who established the anatomic basis for the so-called "radical cure" of hernia and changed almost certain recurrence after herniotomy to predictable success. To a story of slow and

CHAPTER 2

Wound Healing and the Care of Wounds

The function of the surgeon is to establish and [illegible] *for the healing of the wound* [illegible] *Harvey.**

THE LOCAL reactions to injury and wound healing are fundamental biologic phenomena which depend upon the ability of the primordial cell to multiply and differentiate, and thus to replace tissues which have become nonviable. In general, the more differentiated a cell, the less is its ability to regenerate. Mesodermal cells show greater regenerative powers than do ectodermal or entodermal cells.

The healing of a wound has been considered as a reactivation of the normal growth process. Except in degree, the reparative process is essentially the same, whether the wound is accidental or surgical, clean or infected. The basic pattern of regeneration is similar, whether primary or secondary healing occurs.

Under normal conditions, wound healing proceeds more rapidly in actively growing individuals than in those who have ceased to grow. Other than this, there are no known factors that will accelerate the reparative process beyond the normal rate of clean wound healing. There are, however, many factors which can delay wound healing. In the interests of rapid healing, minimal scarring and early restoration of function, it is imperative that the surgeon recognize and eliminate all inhibiting factors, insofar as possible.

* Harvey, S. C.: The healing of wounds as a biologic phenomenon, Surgery 25:655, 1949.

"preoccupation with the local lesion" or the various technics of surgery.

The present builds upon the past, often without recognizing its debt. It would seem only fair to indicate that the difference between the new surgery and the old is no more than a matter of degree. As major problems of operative surgery are conquered and the biologic sciences accelerate, discovery is pyramided on discovery; and it is natural to give more detailed consideration to the general aspects of surgical disorders and to extend the experimental method to the solution of clinical problems. If there is a unique quality to twentieth-century surgery, it is in the increasing effectiveness of surgical care through fuller understanding of the implications of water-electrolyte balance, the need for restoration or maintenance of effective circulating blood volume, an understanding of the metabolic response to injury, the control of infection by specific antibiotic therapy, the importance of adequate oxygenation and safe anesthesia, the role of nutritional support for the depleted patient and numerous other biologic concepts. Advances of surgical biology have paralleled the continuing development of an expert surgical craftsmanship. The sum total of these is the essence of contemporary surgery.

The *surgical trend* of twentieth-century surgery, one can agree with Churchill, is a trend that cultivates the discipline of mind that is needed to complement and to guide an expert surgical craftsmanship. It establishes the surgeon as a biologist and as a physician in the broad sense of one skilled in the healing art. It encourages him to travel in company with others—the internist, the physiologist, the biochemist—in a common quest for the answer to unsolved problems. It is in no way inconsistent with the humanitarian objectives that are treasured by responsible members of a great profession. There is no basis for conflict between the New Science and the Old Humanism.

To this day, it has ever been thus.

ing a permanent approximation of the wound edges. Contraction results in the obliteration of many vascular channels, and eventually the wound scar becomes pale and flattened.

In summary, wound healing is a normal biologic manifestation of growth which proceeds in two directions simultaneously: the removal of devitalized tissue, and the active regeneration of tissue. The cellular activity of the reparative process is characterized by ameboid movement, mitotic division and maturation of cells that are actively engaged in the fusion of tissues. The phenomenon of wound healing may show many faults and imperfections. It is conditioned at the onset by the type of tissue that is involved, and it may be compromised by general and local factors which impair healing.

Types of Wound Healing

The following description of the various ways in which a wound may heal is of clinical value:

Healing by first intention is illustrated by the healing of a simple surgical incision. It is the simplest type of wound healing. The reparative process is uncomplicated, and minimal scarring results.

Healing by second intention, the method of granulation, occurs in the following instances: (*a*) when wound edges are widely separated; (*b*) on exposed tissues when there has been a considerable loss of tissue by trauma or infection, and (*c*) in the body cavities.

Healing by third intention or secondary suture involves the late approximation of a granulating wound surface by sutures, usually after the failure of primary suture.

Delayed primary suture is to be contrasted with so-called "secondary closure." The contaminated traumatic or surgical wound may be left open three to ten days and then closed by suture.

Factors Which Delay Wound Healing

The treatment of wounds requires vigorous care in regard to the general condition of the patient. It also demands meticulous operative technic. The general and local factors which delay wound healing do so by prolonging the time required for removal of nonviable tissue or by preventing or retarding tissue regeneration, or both.

General Factors.—The process of wound healing has priority over many other body functions and usually proceeds, although at a

Normal Wound Healing

The sequence of events in normal wound healing occurs in four stages, although there is considerable overlap of the processes.

Stage of Hemorrhage.—Immediately after injury there is formation of a coagulum which occupies the defect in the tissues resulting from the trauma. The clot consists of a fibrin framework, in which are enmeshed the various blood elements.

Stage of Hyperemia.—This phase is characterized by a vascular and cellular response, due to chemical substances in the tissue juice, and possibly to oxygen lack. There is an increase in blood flow to the area from marked arteriolar dilatation. Lymphatic flow is also accelerated, and various blood elements and wandering cells invade the area of traumatic inflammation. Phagocytosis of bacteria and enzymatic digestion of devitalized tissues begin. There is exudation through the capillary membrane in the zone of injury, but increased lymph flow may remove much of the excess interstitial fluid. The lymphatic system is the chief route for removal of phagocytosed bacteria, foreign bodies and dead tissue. Large accumulations of such material may drain externally as pus if the wound is open.

Stage of Granulation.—As early as the third day of wound healing, vascular sprouts are present. The proliferation of capillaries makes the wound red and exuberant, this is called *granulation tissue*, or "proud flesh." Fibroblasts grow through and along the fibrin network of the coagulum Before the appearance of fibroblasts, the wound has no tensile strength other than that which is contributed by such external means as sutures, splints and pressure. This interval in wound healing, before fibroplasia and collagen formation occur, is called the *lag phase;* in a clean wound, its duration is four to six days. The tensile strength of the wound increases after this according to the amount of fibroplasia and collagen formation but does not reach a maximum for ten to fourteen days after injury. At this phase of the reparative process, autolysis, the active removal of nonviable debris and tissue regeneration proceed simultaneously

Stage of Contraction.—The surfaces of the wound are then covered by the ameboid movement of epithelial cells from the wound margins. The final stage of the healing process may continue for an indefinite period—often for as long as a year. It is characterized by contraction of the wound in length, width and depth, owing to the maturation of cellular elements that have bridged the gap, thus effect-

tight bandages, etc.), may result in partial or complete ischemia with devitalization of tissues. Severe anemia also slows wound healing.

5. Foreign bodies in the wound increase exudation and delay the reparative process. The foreign body may be a metallic fragment or other debris, an excessive amount of suture material, or dead tissue, as, for example, from the use of a mass ligature.
6. Desiccation of tissues from prolonged exposure to air results in devitalization of cells and retardation of wound healing.
7. Improper approximation of tissues, unobliterated tissue spaces and closure of tissues under tension increase the lag phase of wound healing.
8. Neoplastic tissue does not heal well, chiefly because local circulation is deficient.

Classification of Wounds

Classified as to etiology, wounds are due to mechanical, chemical, thermal (Fig. 1), bacterial or other trauma. Listed according to type, there is the abrasion, the laceration, the contusion and the penetrating injury, the wound puncture and the surgical incision. A clinical classification, which is most helpful in its therapeutic implications, follows:

The Clean Wound.—A wound made under aseptic conditions (e.g., the planned surgical incision through a clean field) in which bacterial contamination should not occur.

The Contaminated Wound.—An open traumatic wound that is less than six or eight hours old may be considered to be contaminated; however, the presence of bacteria in an otherwise clean wound does not imply the presence of wound infection. The time interval represents the "lag period" of bacterial growth which precedes actual tissue invasion by bacteria. A contaminated surgical wound is one in which the incision is made through tissues containing pathogenic organisms. Such is the case, for example, in all operations in the mouth.

The Infected Wound.—An infected wound is one in which bacterial growth is occurring. The criteria of wound infection are: (*a*) local changes in the wound, necrosis or suppuration, cellulitis and (*b*) systemic toxicity as manifested by leukocytosis and elevation of the body temperature. Open traumatic wounds of more than eight hours' duration should generally be regarded as infected. A surgical in-

slower rate, in the presence of severe illness and marked deficiency states.

1. The age of the patient is a factor, and wound healing progresses more rapidly in the young than in the old.
2. The nutritional state of the patient, particularly with regard to protein intake, is of the utmost importance. Serious protein deficiency with hypoproteinemia and edema predisposes to prolonged and complicated wound healing.
3. Vitamin C is necessary for the formation of intercellular substance and the maturation of precollagen into the collagen of connective tissue.
4. Vitamin K deficiency with hypoprothrombinemia and a bleeding tendency may interfere with normal wound healing by the formation of hematomas and serum collections. These in turn predispose to wound separation and wound infections.
5. Deficiencies of vitamins A, D, C, thiamine, riboflavin and pantothenic acid lower the rate of phagocytosis and bacterial digestion, thus predisposing to local wound infection.
6. Fluid and electrolyte balance is necessary for optimum wound healing. Dehydration may be associated with prolongation of the lag phase of wound healing. Wounds also heal poorly in the presence of a water-salt overload and edema.

LOCAL FACTORS.—Factors which affect the local conditions in the wound and delay healing include the following:

1. Bacterial infection, either invasive or noninvasive, interferes with the healing process.
2. Devitalized tissue from mechanical or chemical trauma increases the autocatalytic phase of wound healing and also provides a rich pablum for bacterial growth. Antiseptic agents exert no beneficial effect on wound healing, and most antiseptics have more deleterious effects on tissues than on bacteria
3. Hematomas and serum collections prolong the lag phase of wound healing and provide an excellent nutrient media for bacterial growth.
4. An inadequate local blood supply from any cause, including excessive pressure in or on the wound (tissue tension from serum collections, hematomas or edema, wound closure with tension,

1. *Shaving the hair and carefully washing the skin over the* operative site and surrounding area should be done, if possible, several hours before, or the night before, the operation. Liberal amounts of detergent soap (hexachlorophene type) and water should be used. A film of soap should remain on the skin to effect prolonged bacteriostasis.
2. Washing with detergent soap and water should be repeated immediately before phase 3.
3. An antiseptic solution should be "painted" on the operative field and allowed to dry. The local preparation is then complete.

   Several satisfactory antiseptic solutions are available. Certain quarternary ammonium compounds (e.g., Zephiran®) currently are widely used for skin preparation.

During the time the wound is open, the skin margins must be excluded from the wound in order to prevent introduction of any surface bacteria which may remain after skin preparation. Great care must be exercised to prevent drying of tissue, injury from excessive retraction and undue compression or chemical or thermal irritation. The tissues should be divided with sharp instruments, and hemostasis must be meticulous. The wound should contain no free or clotted blood, devitalized tissue or excessive amounts of foreign material (e.g., *ligatures) at the time of closure. Wound cleansing is usually* accomplished by irrigation with large amounts of isotonic salt solution.

The wound is closed by approximating like tissues with sutures. There must be no undue tissue tension; yet all dead spaces must be obliterated. This may require sutures at several levels in the wound, depending on the depth of the wound. All ligatures and sutures should be cut close to the knot, and no excess tissue should be allowed to remain beyond the knot. Simple interrupted sutures and ligatures tied in a square knot are adequate for most purposes. Because there is some tendency for all knots to slip slightly, when maximum security of the knot is desired, three knots, all tied square, are used. Transfixion ligatures (ligatures anchored by sutures) are recommended for the closure of most larger vessels In general, the finest caliber and the least amount of suture material necessary should be used. Thus, the tensile strength of the suture material should not greatly exceed the tensile strength of the tissues in which the sutures are placed.

Two basic types of *material are used as sutures* and *ligatures*—absorbable and nonabsorbable.

Absorbable (plain and chromic catgut).—Catgut is manufactured

cision which traverses or extends into a zone of infection must also be classified under this heading.

Management of Clean Wounds

The best example of a clean wound is that produced by surgical incision through healthy tissues under aseptic conditions. Nearly all other wounds can be considered contaminated or infected.

Surgical incisions should be planned so that they will give ade-

Fig 1.—Injury due to cold (freezing). Note extensive destruction of skin with bullae formation Toes became gangrenous and were amputated after demarcation occurred

quate exposure, will not traverse important structures (e g., nerves and blood vessels), can be closed without difficulty, will be likely to heal without complications and will give the best possible anatomic, functional and cosmetic results.

Before the incision is made, the skin should be treated to minimize or eliminate surface bacteria, in order to decrease the possibility of wound contamination and infection.

The skin preparation should be accomplished in three phases:

Management of the Contaminated Wound

The principal aim in the treatment of the open and contaminated wound is to convert it into a clean closed wound. A faultless, meticulous, aseptic technic is required (Fig. 2). A practical, detailed plan of procedure follows:

1. Place a sterile gauze pad over the wound, shave the surrounding skin, then clean to the edges of the wound with a detergent soap and water. Antiseptic solutions may be used on the operative field, but none should be allowed to enter the wound.
2. Surround and isolate the area with sterile drapes.
3. Remove the dressing which covers the wound and prepare the wound for surgical débridement by gentle irrigation with large volumes of warm isotonic saline solution. The more jagged and irregular the wound, the more painstaking and prolonged the cleansing should be.
4. Do the necessary débridement, removing devitalized and ragged skin edges by sharp dissection (Fig. 3). Nonviable, heavily contaminated and "sure to die" tissue must be excised. Devitalized tissue is usually discolored, does not bleed, and, if muscle, fails to contract on stimulation.
5. Expose every recess of the wound by gentle retraction and carefully irrigate it again.
6. Remove the water-saturated drapes, change surgical gloves, place a sterile gauze pad over the wound and prepare the operative field again.
7. Redrape the area.
8. Ligate bleeding vessels and cut the ties close to the knots. Control capillary and venous oozing by gentle pressure with moist sponges.

The method of wound closure or covering must depend on the local situation. Several alternatives are available:

1. The wound may be closed by suture without drainage (Fig. 4), using fine, interrupted absorbable or nonabsorbable sutures according to the preference of the operator.
2. Primary closure may be done over a small rubber drain placed into the depths of the wound and brought out through the wound margin.
3. The wound may be packed loosely with petrolatum-impreg-

from the submucosal layer of sheep's intestine. Chromic catgut is catgut which has been treated with chromic acid to increase its resistance to the lytic action of cells and body fluids. All catgut produces a local inflammatory reaction in tissues, and is ultimately digested and absorbed Both plain and chromic catgut are available in a variety of sizes and tensile strengths. The sizes most often used in general surgery are: Nos 3–0 (three zero), 2–0 and 1–0.

Nonabsorbable (silk, cotton, Nylon, linen, stainless-steel wire and tantalum wire).—These substances are not dissolved; they remain in the deep tissues permanently. They evoke only a slight inflammatory response, but in the presence of infection they may cause prolonged wound drainage until they are extruded or removed. Silk, cotton and stainless-steel wire are most commonly used. Nonabsorbable materials are also available in a wide range of sizes and tensile strengths. For most operations, silk or cotton Nos. 4–0, 3–0 and 2–0 are used. No. 2–0 is larger and stronger than No. 4–0. Wire sutures are more difficult to handle than the soft materials but have the advantages of less tissue reaction and greater tensile strength.

The choice of suture material depends on the character of the wound and, to some degree, the preference of the operator. Nonabsorbable materials can be used in clean surgical wounds; either absorbable or nonabsorbable materials may be used in contaminated wounds; and, in general, absorbable materials only should be used in infected wounds. As a rule, when the likelihood of wound infection exists, absorbable materials should be used. An important exception to this rule pertains to the closure of certain contaminated or infected incisions with stainless-steel wire because the rapid loss of tensile strength of catgut in the presence of any exudate would make late separation of such a wound a distinct possibility. For example, wire sutures would be preferred when reoperation is undertaken through a recent infected abdominal incision.

The skin margins of the wound are closed with removable sutures, usually of the nonabsorbable type. Simple interrupted sutures, placed close together and tied without tension, give best results. Skin sutures are usually removed on the seventh or eighth postoperative day, except in face or neck wounds, in which case they are removed earlier. In the latter locations, rapid wound healing is the rule and the final appearance of the wound is important, hence, early removal of skin sutures (three to five days) is advisable.

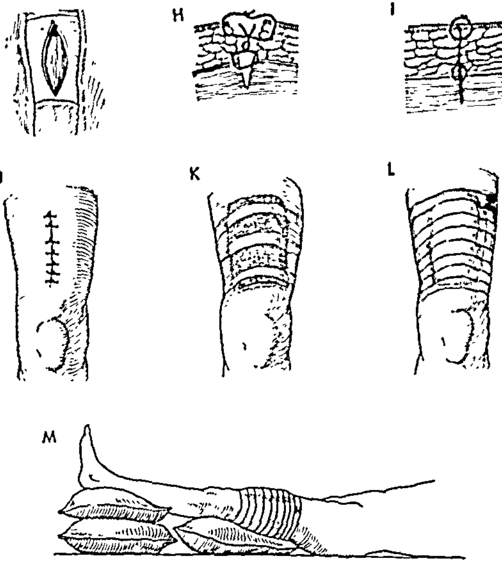

FIG 2 (*cont.*).—*G*, area redraped with sterile towels. Gloves should be changed. *H*, wound closure in layers with fine suture material. *I*, closure of like structures without tension. *J*, external appearance of wound after closure. *K*, sterile dressing applied *L*, slight compression to wound with a circular elastic bandage. *M*, patient placed at best rest with leg elevated

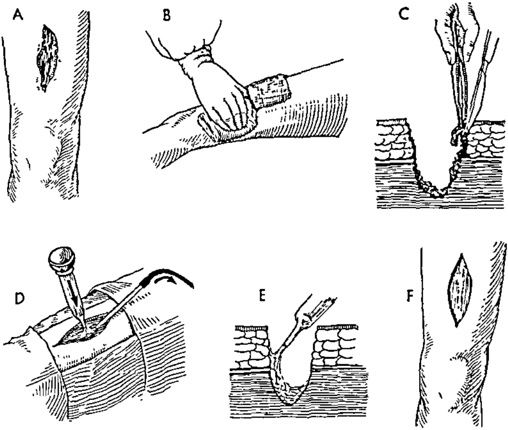

FIG 2 —Steps in local treatment of a traumatic wound of short duration Aseptic conditions throughout *A*, appearance of wound, deep, irregular, gaping and contaminated with bacteria. *B*, wound covered with sterile gauze. Surrounding area of skin cleansed and prepared with antiseptic solution Sterile towels applied to wound margins *C*, wound trimming (débridement), usually in conjunction with wound irrigation *D*, wound irrigation, using copious amounts of isotonic saline solution at body temperature. Simultaneous suction-aspiration is helpful *E*, final irrigation, preparatory to wound closure *F*, appearance of wound at completion of cleansing-trimming

nated gauze or similar material and sutured at a later date (delayed primary closure).

4. If the wound is one in which there is loss of tissue, a skin dressing in the form of a split-thickness skin graft may be used to close the wound

If the wound is traumatic in origin, is grossly contaminated or is of several hours' duration, consider carefully the added risk of infection from primary wound closure. Such a procedure invites aerobic and anaerobic infection. While the antibiotics may to some degree make infection less likely and cover certain deficiencies in treatment, they do

not lessen the need for sound clinical judgment and the application of fundamental principles to the management of wounds.

Care of the Infected Wound

The principles of treatment of infected wounds are:

Localization of the infection
Drainage of the infection when localized, if necessary
Sterilization of the infected area
Covering of the wound surface
Early restoration of function

Some practical points in the application of these principles are:

1. In order to avoid tension, to aid in the localization of infection and to promote drainage, do not close or "plug" an infected wound. Physiologic rest of the affected part aids in localizing the infection.
2. Dependent drainage of wound exudate should be provided, if possible, remembering that pus, like water, will not run uphill. Elevation of the part will aid venous and lymphatic flow, minimize edema and relieve pain that is caused by swelling.
3. Heat in the form of warm, wet packs promotes an increased blood flow to the local area and facilitates drainage of exudates. Moist heat can be maintained for long periods of time by adding sterile, warm, isotonic saline solution to the packs every three to four hours. It is advisable to alternate dry heat and warm packs so as to avoid maceration of tissues.

   Approximately isotonic saline solution can be prepared by adding 2 teaspoonfuls of table salt to a quart of water.
4. Cold applications cause peripheral vasoconstriction with diminution in blood flow to the local area, a decrease in tissue metabolism and bacterial growth and relief of pain. The use of cold is indicated in certain instances:
   a) Very early in the course of an inflammatory process, during the stage of hyperemia, as in the early treatment of a sprained ankle, "black eye," etc.
   b) In the treatment of a cellulitis of the neck (e.g., Ludwig's angina), when danger of airway obstruction is *imminent* and heat may increase the local swelling.
   c) Refrigeration therapy is sometimes used in patients with arterial insufficiency with pregangrenous or gangrenous

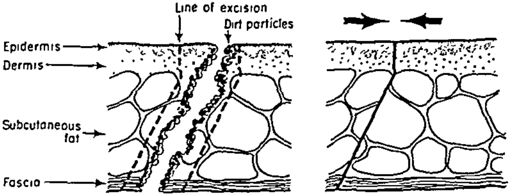

FIG 3.–Principle o'
nated laceration *Left*, e·
in which the tissues are
rately apposed to like tissues. Such meticulous wound healing is required especially for lacerations of the face (From Huffman, W C, and Lierle, D M.: Treatment of facial lacerations, J Iowa M. Soc 42.141, April, 1952)

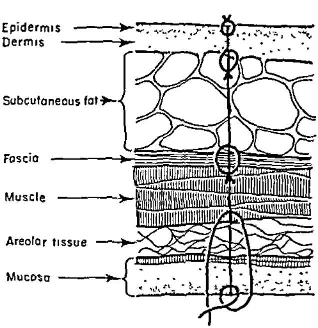

FIG. 4 –Principle of wound closure in layers. Diagram depicts full-thickness laceration of the cheek and sutures in four layers of tissue Beginning at the bottom, the mucosa is held by sutures, the fascial layer is sutured to obliterate the "dead space" and provide strength to the closure, the dermis is sutured to prevent tension on the skin margins, and the epidermis is accurately closed to give minimal visible scarring.

ing of the nature of the infectious process and a knowledge of surgical anatomy and the paths of possible extension from the area involved are basic to the intelligent use of surgical drainage.

The following principles should be observed:

1. Do no harm when establishing surgical drainage.
2. Do not devitalize tissues and impair the blood supply of inflamed tissues by infiltrating them with procaine solution or spraying them with a freezing anesthetic (e.g., ethyl chloride). Regional anesthesia for infections of the extremities is often satisfactory, but general anesthesia is usually preferable.
3. The aseptic technic is mandatory. Prepare the operative field in the manner previously described.
4. Good lighting and adequate exposure are necessary. A tourniquet will aid in securing a bloodless field if the abscess involves an extremity. A blood-pressure cuff inflated above the level of systolic blood pressure is a satisfactory tourniquet. The dangers related to the use of tourniquets, including nerve and vessel injury, should be kept in mind.
5. Do not damage tissues by blunt dissection, forceful retraction or the use of crushing forceps. Use sharp and fine instruments.
6. Make the incision large enough to provide drainage. It should remain open until the infection subsides. It is a common error to make the first incision too small and then to have to subject the patient to subsequent drainage operations.
7. Place the incision through the most direct route possible and preferably in a dependent location, so that gravity will aid drainage of the exudate.
8. Keep the incision within the zone of reaction; avoid cutting into noninfected areas Do not damage important structures.
9. Soft, rubber-dam material or petrolatum-impregnated gauze can be used to keep the wound edges apart. A drain is useful only as long as it is necessary for the escape of exudate; when it no longer serves this purpose, it is only a foreign body in the wound and, therefore, harmful. Usually a drain may be removed after twenty-four to forty-eight hours. Once the drain is removed, it should not be replaced. Do not use through-and-through drainage. Do not place drains in contact with tendons or other structures with a precarious blood supply. They may cause pressure necrosis.
10. Maintain "clean surgical care" through the course of treat-

changes of the leg. Cooling, rather than actual freezing, is utilized unless the extremity is soon to be amputated. Cooling of an infected gangrenous extremity in preparation for amputation will reduce systemic toxicity from infection and give relief from the ischemic pain of arterial insufficiency. In patients with severe diabetic acidosis and gangrene, temporary refrigeration of the extremity may be lifesaving.

Flannel-covered ice bags applied to an extremity often provide satisfactory cooling. The use of a tourniquet is generally unnecessary and undesirable. If the extremity is obviously gangrenous or otherwise not salvageable, it may be packed in ice to a level several inches *below* the site of proposed amputation.

5. The wound dressing should be changed as often as indicated. Instrument dressing changes by masked personnel will reduce the incidence of cross bacterial contamination. Remove necrotic tissue from the wound by excision, and remove exudate by irrigation Do not allow pus to pool in the wound.
6. The more severe the infection, the more caution should be exercised in operating to establish surgical drainage. It is a good clinical rule to make certain that localization of the acute, spreading infection has occurred—then wait a little longer.
7. Four specific types of acute, spreading infection demand nonoperative treatment because of the danger of producing extension of the infection by ill-considered surgical incisions:
   a) Acute lymphangitis and acute cellulitis
   b) Acute thrombophlebitis
   c) Acute infections about the teeth
   d) Infections in the so-called "dangerous area" of the face *

Principles of Surgical Drainage

Surgical drainage becomes necessary if there is a localized collection of pus, or increasing tissue tension threatens to destroy important structures. The signs of localization are (1) "pointing" of the infection, (2) fluctuation and (3) localized pain and tenderness. An understand-

* The "dangerous area" of the face is a triangular zone bounded by the bridge of the nose and the corners of the mouth Infections of this area may extend via the angular veins to the cavernous sinus, producing the serious complication of cavernous sinus thrombosis.

frequently infection are the expected sequelae of undue tissue tension.

9. The principle of immobilization and rest must be constantly re-emphasized in the treatment of soft-tissue wounds, as it is in fractures.

10. The question of closure of wounds of more than eight hours'

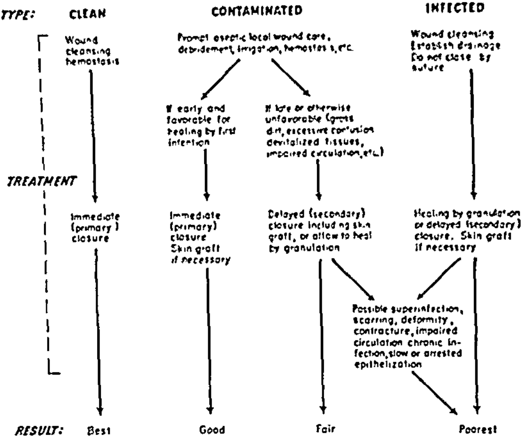

FIG. 5 –Classification of traumatic wounds into categories and correlation with treatment involves consideration of many factors, including the manner in which the wound was produced and the circumstances surrounding its production, the elapsed time between the injury and definitive treatment, and local conditions in the wound. In addition, the condition of the patient must be considered in the therapeutic approach.

duration must be answered on the basis of local conditions in the wound. Under conditions favorable for bacterial growth, invasive infections can develop during this short interval. It is believed that the use of antibiotics will permit some extension of this time with respect to primary wound closure, but the principles upon which treatment must be based are the same, regardless of the use of these drugs.

11. There has been a noticeable general tendency toward laxity

ment of the infection. Avoid superinfection and reinfection by attention to the details of aseptic technic.

11. During the acute phase, keep the affected part at rest. When dealing with hand infections, maintain the hand in the so-called *position of function.*

    The position of function for the hand (Fig. 23, p. 155) is with the wrist dorsiflexed at 45 degrees, the fingers held slightly flexed at all joints, the fingers slightly abducted. The thumb is abducted from the hand and rotated forward, so that its volar surface faces the palm.

12. When the acute inflammatory process has subsided and the infection is under control, institute measures necessary for prompt coverage of the defect (skin grafting may be required) and early restoration of function.

Summary of Some Principles of Wound Treatment

1 Wound healing is a biologic process incited by tissue injury and directed toward the re-establishment of tissue continuity and restoration of function.

2 In wound treatment the primary objective of the surgeon is to establish and maintain those conditions which are essential for rapid and complete healing (Fig. 5).

3. In general, the greater the extent of tissue injury, the greater the delay in wound healing (lytic or lag phase).

4. Likewise, the greater the delay in wound healing, the greater will be the residual imperfections in tissue structure and function.

5. A good blood supply to the tissues in the wound is all-important for healing. Every effort must be made to maintain adequate fluid interchange in the arterial, venous, lymphatic and intercellular components of the circulation.

6 Edema always exerts a deleterious effect on healing. It may be minimized or prevented by proper immobilization, rest, elevation and, in some instances, physiologic degrees of external compression.

7. Foreign bodies (including blood clots, dead tissue, bacteria, debris, sutures, metallic objects) in the wound contribute to increased reaction and delayed healing. Every effort must be made to minimize these deleterious influences.

8. Healing tissues do not tolerate unusual degrees of internal or external tension. Vascular impairment, edema, ischemia, necrosis and

A compensating continuous regeneration in the stratum germinativum keeps pace with this process. In general, epithelial cells have a great capacity for regeneration, which occurs by mitotic division and ameboid movement. Defects are covered by the ameboid movement of the cells bordering the defect, as well as by new cells and contraction of the wound. A thin continuous sheet of epithelium is thus extended over the defect. Regeneration may also occur by metaplasia of cells of accessory skin structures, such as hair follicles and sweat and sebaceous glands. As compared to many other tissues, epithelial regeneration tends to be more rapid.

MUCOUS MEMBRANE.—The remarks already made in connection with the epithelium of the skin also pertain to mucous membrane. The columnar epithelium of the stomach and intestine regenerates from undifferentiated epithelium located in the base of the gastric foveolae or, in the case of the small bowel, in the crypts of Lieberkühn. Mucous membrane regeneration tends to be rapid and differentiation excellent.

MESOTHELIUM.—The serous cavities are lined with a thin, pavement-like layer of flattened cells which in some places appears stratified. Superficially, these cells resemble squamous epithelial cells. Following injury, multifocal transformation of exposed connective tissue cells results in rapid regeneration of the mesothelial surface. Regeneration does not occur from the margins of the defect, as in an epithelial surface. Mesothelial regeneration is generally rapid and complete.

MUSCLE.—It is possible that smooth-muscle regeneration occurs in the uterus during pregnancy from connective-tissue cells of the myometrium, while smooth-muscle cells in the adult retain the ability to divide by mitosis to a limited degree. Smooth-muscle defects generally heal by the formation of scar tissue.

The capacity of striated muscle to form functioning muscle fibers is slight. Abortive attempts at regeneration give rise to bizarre giant cells which may be mistaken for tumor cells. Defects in striated muscle are replaced by a fibrous tissue.

The regenerative capacity of cardiac muscle is even less than that of skeletal muscle.

CONNECTIVE TISSUE.—At the danger of oversimplification, it may be stated that connective tissue has great capacity for regeneration. Depending on the density and specialized function of the tissue, healing may be rapid or slow. Highly specialized connective tissues heal by scar or by regeneration in whole or in part of the antecedent structures. The fibroblast is the parent cell of all connective-tissue repair.

in wound handling and a decreased awareness of the importance of the aseptic technic since the antibiotics have become available. This is an inexcusable attitude, and the student is urged to develop an "aseptic conscience."

12. Wounds of the head and face require special care. The exceptionally good local blood supply in these areas is usually conducive to primary healing. All efforts should be directed toward achieving the least possible scarring and deformity.

13. Multiple injuries are common. Consider and examine the whole patient. Do not be trapped into the error of believing that the single obvious injury is necessarily the most important.

14. Treatment of injuries must be carried out under proper conditions, and, except for those of minor extent, this can be achieved only in a well-equipped hospital.

15. The following details of wound treatment are basic: aseptic conditions, proper skin preparation, careful débridement and trimming, irrigation of the wound with large amounts of saline solution, removal of foreign bodies and debris, careful hemostasis, layer closure, using fine nonirritating sutures, and avoidance of tissue tension.

16. Shock and hemorrhage have priority in treatment. Antibiotics and prophylactic inoculations should be administered early and in adequate dosage. Rest, immobilization and elevation are important postoperative considerations.

17. Remember that restoration of function is the ultimate objective. Rehabilitative measures, including exercises and physiotherapy, should be instituted as soon as they will do no harm.

Healing of Special Tissues

Skin.—The capacity (i.e., ability) for cellular regeneration, the rate of regeneration and the type or means by which repair is attained are of day-to-day interest. These factors have much to do with the "liberties" which may be taken with tissues in terms of surgical technic or end results. In the complex mixtures of tissues of an individual organ, the ability to regenerate varies according to the tissues. In the skin, for instance, repair of the epithelium is different from that of the underlying connective tissue.

Epithelial structures are in a constant state of shedding, degeneration and regeneration. This is true of the epidermis, where the superficial cells are continually undergoing cornification and desquamation

A compensating continuous regeneration in the stratum germinativum keeps pace with this process. In general, epithelial cells have a great capacity for regeneration, which occurs by mitotic division and ameboid movement. Defects are covered by the ameboid movement of the cells bordering the defect, as well as by new cells and contraction of the wound. A thin continuous sheet of epithelium is thus extended over the defect. Regeneration may also occur by metaplasia of cells of accessory skin structures, such as hair follicles and sweat and sebaceous glands. As compared to many other tissues, epithelial regeneration tends to be more rapid.

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A severed tendon may fail to heal because of separation of the cut ends. The tendon receives a precarious blood supply through a specialized mesentery-like vascular connection known as the "mesotendon." If the tendon ends are held in close apposition during healing (Fig. 6) and the blood supply is not seriously compromised, the two cut ends may be united first by a fibrin clot, then by granulation tissue and finally by scar tissue. If healing occurs with minimal scarring, function may be restored. If, however, a gap exists between the severed ends and scar-tissue formation is excessive, or muscle pull causes stretching of the scar, a functionless tendon may result.

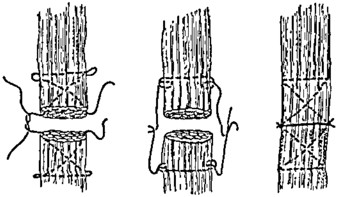

FIG. 6 —Principles of tendon suture Accurate union of the divided tendon ends with nonabsorbable (silk, cotton, wire, etc.) material. Note that the suture knots must not come between the apposed ends of the divided tendon. *Left and center*, methods of anchoring the tendon ends *Right*, apposition of ends after sutures have been tied.

The dermis also heals by scar. If the healed dermis is subjected to the original mechanical stresses and strains and if injury has not been excessive, the collagen bundles may be reoriented in such manner as to functionally imitate the dermis which existed before the injury. This type of restoration in response to functional stimulation is a characteristic of connective-tissue healing, and the same process of adaptation is seen in the healing of fractures.

Adipose tissue is a form of modified connective tissue in which the cells store neutral fats. Fat cells develop by metaplasia from primitive connective tissue cells Dedifferentiation back to pure connective-tissue cells is possible upon release of the stored fat. Regeneration of the adipose tissue is possible in the sense that additional adipose-tissue cells are formed at the site of an initial or original deposit of fat after an injury. However, wounds across adipose tissue heal by scar.

NERVE.—Neurons, once destroyed, are not replaced; but nerve fibers have a striking capacity for regeneration. This is in contrast to the very limited regeneration of nerve cells and their processes within the brain and spinal cord.

Peripheral nerve injuries may be grouped according to Seddon's classification. (It should be pointed out that transitions between these categories exist.)

Grade I (neurapraxia): temporary injury with rapid recovery.
Grade II (axonotmesis): destructive injury of axons but not of the supporting matrix. Axons regenerate with good functional recovery.
Grade III (neurotmesis): destruction of all parts of the nerve. There is little likelihood of functional return unless anatomic restoration of the cut ends is established.

In Grade I nerve injury, there is no axonal degeneration but there may be slight myelin degeneration. Contusion, especially to heavily myelinated nerves conveying motor and deep sensation, is the common cause. Spontaneous recovery occurs rapidly in a few days or weeks.

In Grade II injury, there is a local disruption and destruction of the nerve fibers (axis cylinder and myelin). The continuity of the nerve is preserved and the endoneurial and Schwann cell sheaths are not destroyed or disrupted. Degenerative changes occur in the axon and myelin sheath distal to the injury. Variable degrees of degeneration also appear proximal to the nerve injury. Generally, the farther the injury is along the course of the nerve fiber, the less will be the extent of proximal degeneration. When the injury to the nerve fiber occurs close to the cell body, the cell may die. After a Grade II injury, there is a latent period of about three weeks before the proximal stump of the axon begins to grow down the sheath. During the latent period, there is proliferation of Schwann cells along the extent of the disintegrating axon and myelin sheath. Phagocytosis of the degenerated sheath elements occurs. The regenerating axons now grow down through a tube of proliferating Schwann cells. Remyelinization precedes functional recovery. In myelinated nerve fibers the regenerating growing tip is at first unmyelinated.

Recovery proceeds at a fairly well-determined rate. The old rule of growth of "1 mm. a day" applies to the rate of regeneration of the axon, and the rate is probably slower than evidence of functional recovery would indicate.

In Grade III injuries, there is complete disruption of the nerve fiber, the supporting connective tissue and the Schwann cells. After

such injuries, spontaneous functional recovery cannot be anticipated. Schwann cells grow out of the proximal stump in the first weeks following injury The regenerating axons also grow out in a helter-skelter manner. Ultimately a snarl of nerve fibers, Schwann cells and scar known as an "amputation neuroma" is produced.

If the divided nerve ends are accurately united (see Fig. 7), variable degrees of functional recovery will follow. Surgical approximation cannot insure against misalignment and misdirection of the regenerating fibers. Some fibers may go astray: thus, motor nerve fibers may enter sensory nerves, or motor nerve fibers from one source may

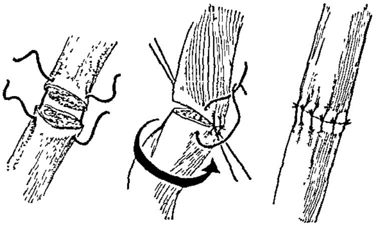

FIG 7.—Principles of nerve suture. Accurate union of divided nerve ends without tension, using fine nonabsorbable material which does not pass through the nerve but incorporates only the nerve sheath. *Left and center*, methods of anchoring the nerve ends *Right*, apposition of ends after sutures have been tied

go into other nerves to new muscles The gap in the nerve is bridged by proliferating Schwann cells and followed by the regenerating axonal processes after a latent period, said to be about seven weeks in man There is a variable amount of branching of axon fibers at the site of nerve injury, which gives rise to more fibers in the distal than the proximal segment of the nerve.

Regeneration of sympathetic nerves is in a different category of potentiality. Preganglionic myelinated fibers may regenerate to an amazing degree and bridge extensive gaps in re-establishing continuity with their ganglia.

CARTILAGE.—Regeneration of hyalin cartilage in the adult does not occur. The defect is filled with scar tissue, which undergoes a limited metaplasia resulting in the formation of some new abortive cartilage.

The new cartilage is usually imperfect, and there is a well-defined residual defect. The repair of cartilaginous defects is generally slow.

BONE.—Bone is a specialized connective tissue consisting of a peculiar matrix which takes up inorganic salts. When fracture occurs, there is disruption of the bone and, to some degree, the periosteum and endosteum. A hematoma which undergoes organization is formed, and then granulation tissue brings in with it the multipotential elements capable of new bone formation. This process is speeded by rapid proliferation of specialized osteoblastic cells which are particularly rich in the inner or cambium layer of the periosteum. The complex mass of precursor tissues seen in about ten days at the site of the fracture hematoma is known as "callus." It consists of proliferating immature cartilage and primitive osteoid tissue; side by side, the transformation proceeds directly or indirectly to bone production. In the indirect method of bone regeneration, cartilage is replaced by osteoid tissue and finally by bone. As osteoid tissue takes up the complex mineral salts, immature bone trabeculae are produced and more and more rigidity of fixation takes place. The immature trabeculae are torn down and replaced, not once but many times; and as each replacement is accomplished, the structure becomes more complex, more compact, and sounder from a mechanical standpoint, until finally adult lamellar bone is produced. The bulky callus slowly diminishes in size, so that by the time healing is complete it can hardly be distinguished from the normal bone.

The role of mechanical forces in the stimulation of fracture healing is not fully understood. Perhaps some movement is desirable; in fact, it is difficult to attain absolute immobility at a fracture site. There is some difference of opinion with respect to the degree of immobilization which is desirable from a theoretical standpoint.

TRANSPLANTATION OF TISSUES.—The transplantation of tissues presents an ever increasing field of activity in modern surgery. The transplantation of tissues from one site to another in the same individual is the form in which it is ordinarily practiced. Such transplants are known as *isografts* or *autografts* and do not present a serious biologic problem. Likewise, cross-transplantation from one identical twin to the other does not appear to be biologically difficult. But when transplants are made between individuals of the same species (*homografts*), a barrier is met which presents serious obstacles to success. Such transplants may be well tolerated for a time but then evoke a rejection response on the part of the host which results either in necrosis of

the graft or its ejection as an unwelcome foreign body. This phenomenon has many of the characteristics of an antibody-antigen reaction. Successive transplants from the same donor to the same host result in acceleration of the rejection response. For example, when homografts of skin are used, the first transplant may survive for as long as two months, the second for only three weeks, and the third may not "take" at all.

In the past decade there has been a great revival of interest in transplantation of vessels, bone and even of whole organs. Vessel and bone homografts are successfully used as temporary stents or splints which are ultimately absorbed or replaced by the host tissues.

Zoografting, that is, transfer of tissues between species, is of little practical importance.

Skin grafts are extensively employed to cover cutaneous defects. They may be of "split thickness," including the epidermis and a portion of the dermis, or of "whole thickness," including all of the dermis and epidermis. In general, the thinner the skin transplant, the greater are its chances for survival; but the thicker the transplant, the better the functional and cosmetic result will be.

When a skin transplant is placed on a recipient bed, which may be either granulation tissue or raw (nonepithelial) tissue, it is at first not attached to the underlying surface. Then fibrin, which is deposited between the bed and the transplant, binds the surfaces by conglutination. Maximal conglutination is usually attained in about seven hours Until the graft is anchored by ingrowing blood vessels and collagen strands (as a result of fibroblastic proliferation), it is in the parasitic phase of transplant existence. In skin, this is usually about forty-eight hours.

Capillaries from the recipient bed invade the transplant, and ultimately it becomes revascularized. Much of the capillary ingrowth is willy-nilly and appears purposeless. Some of the new vessels grow into old vessels and some rupture into venules, filling them with poorly oxygenated blood. This may give the transplant a cyanotic appearance.

Fibroblastic proliferation continues apace with capillary ingrowth, and collagen fibers soon appear. Likewise, some migratory fixed-tissue wandering cells cross the interphase and appear in the graft.

As revascularization proceeds, necrosis of the transplant not en masse, but in sequential order, occurs and can be seen microscopically. First there is necrosis of the epidermis, extending from the outer to the deeper portions. The lingering necrosis of the dermis is slower

in developing. As necrosis proceeds, proliferation of new cells from the transplant's cells that were last to die occurs. In the epidermis this occurs in the germinal layer. The simultaneous death and replacement of cells goes on more rapidly, but otherwise in a manner similar to that which normally occurs in most tissues all the time. This process has been called "creeping substitution."

Later, other important changes occur, which constitute final restitution. These have to do with the adjustment of the transplant to its new surroundings and its response to the inherent mechanical stresses and strains. These forces determine to a considerable degree the pattern of the collagen fibers in the dermis and the thickness and behavior of the epidermis.

Bone grafts are widely used in clinical surgery for the correction of disorders of the skeletal system. Many of the remarks regarding the transplantation of skin apply as well to bone. In general, thin pieces of bone have a better chance of survival or "take" than thick ones. Cancellous bone grafts fare better than cortical bone grafts and are more rapidly incorporated into the host bed.

When a bone graft is laid against a vascular recipient site and mechanical fixation is provided, changes transpire which result in the incorporation of the graft into the adjoining bone by solid bony union. The graft is absorbed and replaced by new living bone simultaneously by "creeping substitution." When bone is transplanted free into soft tissue where it is not subjected to mechanical stresses and strains, it is ultimately absorbed or cast out as a nontolerated foreign body.

After the transplant has been completely replaced by new bone, it still may undergo extensive remodeling, depending on the mechanical stimuli placed upon it.

Tendon grafts, or transplants, with attached paratenon are more successful than transplants of isolated tendon. Accurate approximation of tendon ends and immobilization is necessary for satisfactory healing.

Vascular grafts are widely used to bridge defects in major arteries. Both artery and vein transplants are employed. There are obvious limitations to using arterial autografts, but vein grafts may be used to bridge arterial defects in the same patient. For example, a section of the saphenous vein may be removed and substituted for a segment of the femoral artery. Properly prepared arterial homotransplants, removed soon after death, are well tolerated and serve as inert conduits until they are ultimately replaced by host tissue. Sometimes the

transplant is the site of thrombosis, aneurysmal dilatation or late calcification. Homologous transplants are obtained from appropriate donors at autopsy under aseptic conditions and can be stored in plasma or in special solutions under refrigeration for a few days to several weeks. Various methods of chemical and radiation sterilization of grafts have been used. At present the freeze-drying method (lyophilization) is most widely used for preservation of arterial grafts.

Other tissues which are less frequently transplanted are: muscle, cartilage, fascia, cornea and nerve. Successful transplantation requires unusual care in the selection of suitable subjects and application of proper technics.

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CHAPTER 3

The Systemic Response to Injury

There is a circumstance attending accidental injury which does not belong to disease—namely, that the injury done has in all cases a tendency to produce the disposition and the means of cure.—JOHN HUNTER, 1794.

AS THE SURGEON sees it, injury is usually due to various types of trauma, anesthesia and surgery, infection, obstruction, thrombosis and embolism and new growths. Injury incites both local and systemic responses as survival mechanisms to protect the organism from the damaging effects of the injury. Thus the process of recovery is initiated at the time of wounding. The local changes which occur are specific effects of the different stressors, and each burn, bullet wound, laceration, crush or other severe injury produces its own complex of local damage and complications. There is an individuality about *the local injury,* in any case, that is related to its cause. If the *specific local effects* are overwhelming or uncompensated, they are veritable death mechanisms; for the trauma itself is locally destructive and generally disintegrating, since it damages or destroys cell aggregates or entire organ systems. If the magnitude of the injury is sufficient, the local changes which take place are closely integrated with a variety of systemic responses—the reaction becomes, in fact, a *total body response.* This generalization is effected through both neural and humoral stimuli, since innervation in an injured area is not essential to activation of the whole organism and the infinitely complex metabolic-endocrine reaction in which the central nervous system, the hypothalamus and the pituitary-adrenal axis play key roles.

For the purposes of this discussion, attention will be focused on the systemic responses to injury. The unique and characteristic features of the local responses to injury were discussed in Chapter 2, on Wound Healing, and will also be considered in Chapter 7, on Surgical Infections and Their Treatment. It should be emphasized, however, that this is an artificial separation and that the normal pattern of convalescence can be understood only in the light of the totality of the body responses, local and general.

The systemic response to injury has been subjected to intense study in recent years. The development of the stress concept and description of the general adaptation syndrome (Selye) and data from numerous metabolic studies of the neuroendocrine response to stress and surgery in experimental animals and the surgical patient, utilizing isotopic technics and controlled balance study on the postsurgical metabolic wards, as well as many other clinical and laboratory observations, have made possible the formulation of "concept of convalescence" that is of the utmost practical value in the care of surgical patients. It is to be noted that this synthesis is based on fragmentary information and future research may very well require that modifications of it will be necessary.

The biologic forces activated by the total stress of surgery are concerned with:

1. The problems of survival during the course of the emergency. Circulatory homeostasis and energy requirements of the organism have first priority during this period.
2. Wound healing.
3. Restoration of homeostasis, a return to the zero balance of normality and health.

In order to illustrate the systemic response to injury, consider the clinical changes which may be observed when an otherwise healthy young woman is subjected to cholecystectomy for uncomplicated chronic gallbladder disease. Assume that the patient is in good general physical condition, that the operation is performed under general anesthesia and that it is attended by no unusual trauma, blood loss or other stresses. The immediate postoperative course is smooth, the wound heals by primary intention, and the patient is discharged from the hospital on the ninth postoperative day. The patient continues to improve for about six weeks, during which time she regains her former weight and strength and recovers from the effects of operation.

During the preoperative phase, the patient's physical condition

is studied with a view to assessing the surgical risk, as well as to the detection of deficiencies that might exist and their correction. The cardiovascular, pulmonary and renal function evaluations are within normal limits. Blood values are normal, and fluid balance is not disturbed. The patient exhibits no untoward emotional reactions. There is no indication that her homeostatic reserves are impaired, and a good response to surgery is predicted. In other words, she is considered to be a good surgical risk. Despite this, whole blood is typed and cross-matched so that it is readily available in case blood loss or some other basis for surgical shock should make transfusions necessary. Since there will be a predictable interference with gastrointestinal function in the postoperative period due to the ileus of laparotomy and intraperitoneal manipulations, no food or fluid is allowed during the immediate preoperative period. As a precautionary measure, a nasogastric tube is inserted just before the patient is taken to the operating room. Preanesthetic medication, including morphine and atropine, has been given to protect further the patient from the stress of anesthesia and operation by causing a decrease in metabolism, a depression of reflex activity, decrease in psychic activity and increased tolerance to pain stimuli.

During anesthesia and operation, the respirations and blood pressure are supported and fluid losses are replaced volume for volume by parenteral therapy. Under these circumstances, there is little change from the preoperative "steady state"

The patient awakens soon after anesthesia is discontinued. She complains of pain in the region of the incision and tends to lie quietly in bed. Her respirations are shallow and more rapid than normal Soon she becomes more responsive and may ask questions. Her color remains good without cyanosis, and pulse and blood pressure, frequently checked, remain stable There is some postanesthetic nausea with retching, but it does not last for long. The patient sleeps intermittently. Later, she complains of increased wound pain. A small amount of opiate is administered, and she sleeps some more.

After six or eight hours, she has a sensation of bladder fulness and urinates with difficulty, because straining causes wound pain The urine volume output is small, only 150–200 ml. Only a little fluid is aspirated from the nasogastric tube, although the tube is kept open by periodic irrigation with isotonic saline solution

During the first night the patient sleeps quietly, aided by small amounts of analgesic drugs. On awakening, she complains of con-

siderable wound pain on movement and wants to be left undisturbed. Her surgeon, however, insists upon early ambulation and helps the patient into the sitting position on the side of the bed. At first there is some dizziness, but her postural hypotension soon disappears as vascular adjustments are made to the shift. There is a perceptible increase in heart rate and a stronger pulse. The patient is then able to stand at the bedside and walk about with assistance. She is grateful to return to bed and states that she has been exhausted by the effort of these first few steps; she then falls asleep. Each successive period of ambulation is better tolerated than the preceding one, and soon the patient is able to be about without assistance.

On the first postoperative day, and for two or three days thereafter, a body temperature elevation of about one degree (F.) is observed. This slight temperature elevation is a part of the metabolic response. Postoperative atelectasis, wound infection, dehydration or other complications, should they occur, will generally cause higher and more prolonged elevations of temperature.

Gastrointestinal function is depressed during the early phase of the postoperative period. The patient has no appetite; she complains of transient nausea, vomits if fluid or food is taken, and passes no gas or feces. The decreased bowel activity is also indicated by the findings on auscultation of the adbomen. Peristaltic sounds are greatly diminished or absent. Likewise, there is decreased secretory activity, as indicated by the small amounts of bile-stained fluid which appear in the suction bottle. If the nasogastric suction is continued, the amount of aspirated fluid will be in the range of 100–300 ml. every twenty-four hours for the first three to five days and will then increase. Under ordinary circumstances, the nasogastric tube is removed after the first or second postoperative day. Appetite and gastrointestinal function return, oral intake is gradually resumed, and parenteral intake is decreased and then discontinued.

The urinary output is decreased to about 500 ml. every twenty-four hours during the immediate postoperative period until a third or fourth day diuresis occurs. Oliguria is associated with decreased sodium and increased potassium excretion. This renal response is due to neuroendocrine influences, antidiuretic hormone (ADH) and adrenocortical hormone (ACH) mechanisms which have been physiologically activated by the stress of surgery. As ADH and ACH withdrawal occur, a water and sodium diuresis takes place and the renal excretion of potassium falls.

Early in the postoperative course the patient exhibits little interest

in the goings-on about her. She complains of weakness and fatigue and prefers to sleep much of the time. Wound pain, which has been severe, becomes less conspicuous; and by the third day, analgesic drugs are no longer needed. Soon the patient is spending most of her waking hours out of bed. She begins to chat with the other patients in the ward, looks ahead to visiting hours, puts on her lipstick and does up her hair, does some light reading and may request a radio or a television set at the bedside.

In the meantime, the wound is undergoing repair. During the initial lag period of wound healing, there is intense cellular activity: inflammation, formation of a fibrin scaffolding and capillary ingrowth. Fibroblastic proliferation and collagen formation begin about the fourth or fifth day, and the wound starts to acquire tensile strength. Wound healing proceeds, so that in seven to nine days the skin margins will remain closed without sutures. A period of four to six weeks is required, however, before significant near maximal tensile strength is gained. During this time, maturation of collagen and contraction of the wound in all dimensions are taking place.

During the height of the postoperative response the patient shows rapid weight loss and there is measurable negative nitrogen balance. This results from the combined effects of starvation and protein catabolism. Only if the period of bed rest is prolonged for weeks does immobilization itself become a factor in this metabolic response. Weight loss may exceed a pound a day. About one half of the weight loss is due to the metabolism of fat stores. As soon as the patient takes oral feedings and her caloric and maintenance protein requirements are met, weight loss ceases. Nitrogen balance, which has been negative, becomes zero, then strongly positive as the patient shifts into the anabolic phase of convalescence. It may require four to six weeks for her to regain preoperative weight. Nutritional restitution is accompanied by a feeling of well-being, increased strength and endurance. The return of reproductive function as manifested by a normal menstrual cycle is usually delayed for several months.

At this stage, the convalescence is complete and recovery may be said to have been achieved.

The Neuroendocrine Response

The systemic response to injury is in large degree initiated and sustained by the activation of neuroendocrine mechanisms, of which

the hypothalamic-pituitary-adrenal system (Fig. 8) is most critical.

NERVOUS DEFENSE MECHANISM.—Following injury, there is a quick mobilization of body resources for the purposes of immediate survival. This is accomplished via the adrenal medulla and the autonomic nervous system with release of the physiologic chemical mediators, epi-

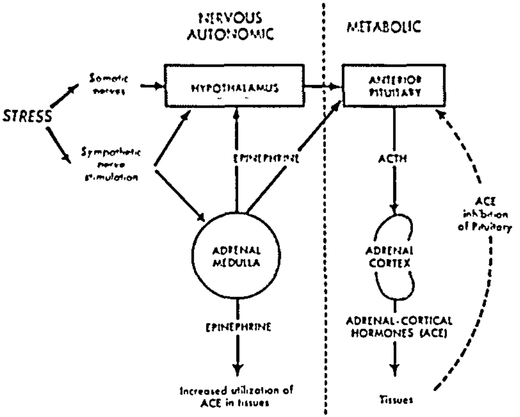

FIG. 8.—The pituitary-adrenal axis which is activated by "stress." Note the role of the adrenal medulla and the hypothalamus in the initiation of reaction to stress. The level of the adrenocortical hormones (ACE or ACH) in the circulation regulates release of adrenocorticotrophic hormone (ACTH) from the anterior pituitary. This is a "feedback" reciprocal control mechanism. (From Richards, V., Surgery of the adrenals, Am. J. Surg. 89:1215, June, 1956.)

nephrine and nor-epinephrine. Activation of this emergency mechanism for "fight or flight" may result from various types of stressors: emotional stress, fear, blood loss and hypotension, increased central nervous system activity, etc. All vital body reactions are instantly mobilized to peak efficiency, while less important organs stop their functions and become relatively ischemic. Adrenergic peripheral vasoconstriction, increased peripheral resistance, increased blood pressure, tachycardia and increased cardiac output result. Circulatory

homeostasis and provision for quick energy expenditure with increased blood flow through lean muscle mass and glycogenolysis are its objectives. Pupillary dilatation and sweating occur as side actions. The adrenomedullary response is prompt but of short duration, lasting only one to twelve hours.

Hormonal Defense Mechanism.—Serum corticoid levels are elevated after a short latent period following injury, anesthesia or surgery, and the stage is set for a relatively prolonged response. One should keep in mind that these responses vary in rate and magnitude, from time to time, and roughly parallel the severity of the stress, and that the range of the so-called "normal" response is quite broad. The essential stimulus may be either neural or hormonal in origin. An afferent barrage from an area of injury, spontaneous central nervous system activity from strong emotions, such as fear, anxiety, or worry, excitement during induction of anesthesia, endogenous or administered epinephrine, and unknown humoral substances, will trigger the hypothalamic-anterior pituitary-ACTH-adrenal cortex. This will be followed by a sudden and sustained increased level of circulating adrenocortical hormones. It would seem that the purpose of this mechanism is to conserve the body fluids, mobilize energy stores, combat shock and, at a later stage in convalescence, aid in the healing of the wound.

The metabolic response to activation of the pituitary-adrenal axis, which can be reproduced by the administration of exogenous ACTH, includes the following features: negative nitrogen balance, eosinopenia, sodium and water retention, potassium diuresis, increased serum corticoid levels and an increased urinary excretion of corticosteroids.

The adrenocortical hormones are classified into three groups, according to their characteristic metabolic effects:

1. *Desoxycorticosterone*-like hormones (DOCA,® aldosterone, mineral corticoids).—Salt and water metabolism are influenced through the renal mechanism of sodium retention and potassium excretion.
2. *Corticosterone* and its derivatives (11-oxysteroids, glucocorticoids).—These hormones affect carbohydrate metabolism by the conversion of exogenous carbohydrate to glycogen, protein to carbohydrate (*gluconeogenesis*); and also cause an *insulin-resistant* hyperglycemia and glycosuria, so-called "postsurgical diabetes." Protein catabolism is associated with increased urinary nitrogen and potassium excretion. There is associated lysis of circu-

lating lymphocytes with increased titer of immune gamma globulin, and a disappearance or depression of the circulating eosinophils. The total eosinophil count correlates inversely with adrenocorticoid secretion and is widely used as a screening test of this endocrine function.

3. *Androgenic hormones.*—Similar to the androgens of testicular origin, measured by 17-ketosteroid excretion in the urine, they are concerned primarily with sexual characteristics and function, although these hormones also exert an anabolic effect on protein metabolism.

Thyroid gland participation in the systemic reaction to injury is probable but not established. The thyroid is under neuroendocrine control, via the elaboration of thyrotropic hormone (TSH) from hypothalamic-pituitary activation. Increased levels of thyroxin heighten the metabolic rate of all tissues.

In uncomplicated convalescence, serum corticoid levels remain elevated for a period of three to five days. Before corticoid withdrawal, the circulating eosinophils are decreased or absent, there is negative nitrogen balance, potassium diuresis, sodium retention and increased urinary excretion of corticosteroids. Corticoid withdrawal is accompanied by a reversal of these changes.

The small available stores of carbohydrate are rapidly exhausted in the initial period following injury, so that protein and fat stores are mobilized to meet the energy requirement. Weight loss and increased utilization of fat stores continue for five to seven days, or until caloric requirements can be met by oral intake. This trend is reversed by corticoid withdrawal and the resumption of oral feedings.

Protein catabolism and negative nitrogen balance occur regularly as a part of the metabolic response, owing in part to gluconeogenesis and in part to starvation. The products of protein breakdown are used in tissue repair, but the prodigal rate of this catabolic process leads to large net losses Again, corticoid withdrawal is associated with decreased urinary nitrogen losses; and, when protein and caloric requirements are met, the nitrogen balance becomes zero, then positive.

There is immediate reduction in urinary and sweat sodium concentrations following the adrenocorticoid response to trauma. Activation of the ADH mechanism by stress potentiates the oliguria which develops. During this period, serum sodium concentrations may be less than normal. This may represent a sodium "shift" from the extracellular fluid to the intracellular space, with sodium displacing potassium. A

homeostasis and provision for quick energy expenditure with increased blood flow through lean muscle mass and glycogenolysis are its objectives. Pupillary dilatation and sweating occur as side actions. The adrenomedullary response is prompt but of short duration, lasting only one to twelve hours.

HORMONAL DEFENSE MECHANISM.—Serum corticoid levels are elevated after a short latent period following injury, anesthesia or surgery, and the stage is set for a relatively prolonged response. One should keep in mind that these responses vary in rate and magnitude, from time to time, and roughly parallel the severity of the stress, and that the range of the so-called "normal" response is quite broad. The essential stimulus may be either neural or hormonal in origin. An afferent barrage from an area of injury, spontaneous central nervous system activity from strong emotions, such as fear, anxiety, or worry, excitement during induction of anesthesia, endogenous or administered epinephrine, and unknown humoral substances, will trigger the hypothalamic-anterior pituitary-ACTH-adrenal cortex. This will be followed by a sudden and sustained increased level of circulating adrenocortical hormones. It would seem that the purpose of this mechanism is to conserve the body fluids, mobilize energy stores, combat shock and, at a later stage in convalescence, aid in the healing of the wound.

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TABLE 1.—CLINICAL-BIOCHEMICAL COURSE OF CONVALESCENCE *

PHASE	CLINICAL	THE WOUND	METABOLIC
Preoperative	Fear and apprehension. Acute starvation with cessation of oral intake		Zero balance of nitrogen, water-electrolyte and blood volume. Eosinophils drop slightly.
	OPERATION		
Neuroendocrine (epinephrine) PHASE I ADRENERGIC-CORTICOID PHASE 1–4 days	Adrenal (emergency) response, 1–12 hours. Listless, inactive, lack of interest. No appetite	No healing. Formation of fibrin coagulum.	Rapid weight loss, from utilization of carbohydrate, fat and protein stores. Negative nitrogen and potassium balance. Retention of sodium and water. Eosinophils disappear. Corticoid excretion increased
PHASE II CORTICOID WITHDRAWAL PHASE 5–8 days after operation	Ambitious but weak. Some activity. Some return of appetite	Fibroplasia, beginning collagen deposition. Wound begins to acquire tensile strength.	Rate of weight loss decreases. Nitrogen loss decreases, potassium balance restored. Lean tissue destruction ceases. Sodium and water diuresis. Caloric intake increases. Eosinophils rise and corticoid excretion falls.
PHASE III SPONTANEOUS ANABOLIC PHASE 9–30 days after operation	Ambitious and stronger. Diet "permits" this phase to start	Tensile strength rapidly increased as wound heals. Inflammation, pain and tenderness disappear.	Weight gain with positive nitrogen balance (3–5 Gm./24 hr. gain). Calories 150/1 ratio to nitrogen or better. Sodium zero balance. Eosinophils and corticoid excretion normal.
PHASE IV. FAT GAIN PHASE *Weeks or months*	Return of full body weight and function, including reproductive	Wound flattens, broadens, whitens	Metabolic balance restored.

* After Moore, F. D. Bodily changes in surgical convalescence. I. The normal sequence—observations and interpretations, Ann. Surg. 137: 289, 1953

sodium-potassium shift in the opposite direction then results, with corticoid withdrawal and a period of salt-and-water diuresis. After about seven to ten days, sodium balance again becomes zero. Renal losses of potassium are increased during adrenocortical stimulation. Potassium release from its predominantly intracellular position may result from cellular breakdown or a sodium-potassium shift. Each gram of nitrogen broken down results in the liberation of 2.7 mEq. of potassium. Under conditions of adequate renal function, potassium excretion is rapid and there is little danger of potassium intoxication. Potassium diuresis ceases following corticoid withdrawal, and balance soon becomes zero, then strongly positive as the resynthesis of tissue occurs in the weight gain phase of the convalescence.

Francis D. Moore has suggested that convalescence consists of four phases (see Table 1), and he has given each phase an appropriate title. Although the total duration of each phase may vary and there is overlapping, the average duration of the sequential phases of convalescence following moderately severe surgical trauma is as follows:

I. Adrenergic-corticoid phase, about five days
II Corticoid withdrawal phase, about four days
III. Spontaneous anabolic phase, about twenty days
IV. Fat-gain phase, about forty days

Therapeutic Corollaries

Following operation or injury, treatment should be provided which meets the patient's changing needs and avoids deficits or overloads. There is a normality of convalescence, a pattern of recovery, which must be understood in order to maintain "balanced" therapy, as well as to understand the abnormalities of response which may develop.

PHASE I. Adrenergic-Corticoid Phase

1. The essential energy and water requirements may be supplied by the administration of glucose in water when only parenteral routes are available

2. Overloads of salt and water should be avoided. Excesses cannot be excreted during the phase of adrenocortical stimulation. Water-salt retention results in edema.

3. Restoration of nitrogen balance cannot be achieved during the height of the adrenocortical response. Parenteral nitrogen will not be

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used to build new protein but will be metabolized to meet the caloric requirement. Urinary nitrogen losses increase, do what you will.

4. Potassium administration during the postoperative period is aimed at balanced replacement. The oral route should be used if available

5. Whole blood in amounts sufficient to replace blood loss should be given as indicated during all phases of the recovery.

PHASE II. CORTICOID WITHDRAWAL PHASE

1. Parenteral fluid therapy can be reduced, then discontinued when gastrointestinal function resumes. Oral intake is started and increased as tolerated.

2. Early ambulation and increasing general activity will counter the adverse effects of immobilization and speed recovery.

3 *Now the local healing process proceeds rapidly and the wound* gains tensile strength. Usually the sutures can be removed at the end of this phase, but continued wound protection is needed because union is still insecure

4. If there are no complications, the patient is able to leave the hospital and continue the convalescence at home.

PHASE III. SPONTANEOUS ANABOLIC PHASE

1. A liberal diet, high in carbohydrates, protein and vitamins is important It permits the anabolic process to begin and to proceed at a rapid pace.

2. Weight gain takes place, and with it comes increased strength, endurance and ability to work. The patient should be encouraged to resume graded activity and social contacts.

3. The wound should be protected against unusual stresses and strains The possibility of a deep separation and hernia formation still exists.

PHASE IV. FAT-GAIN PHASE

1. There is steady weight gain, metabolic balance is restored, and fat stores are reconstituted.

2 The wound unites firmly and the scar becomes less prominent; local pain and tenderness disappear.

3. Sexual function becomes normal.

Convalescence is at an end.

and the kidneys, intestinal tract and the lungs provide the mechanisms needed for the discharge of the waste products of metabolism. These routes for intake and output make possible the chemical reactions, such as synthesis and degradation, as well as the energy production required to maintain the individual at a more or less constant level. In order for these reactions to occur, it is necessary that dilution or concentration of the substances entering into the reaction, the pH of the reaction and the temperature be within a narrow range of variation. When great variation occurs, the chemical reactions become inadequate and imbalance or illness results. The constancy of the internal environment is, therefore, of paramount importance for the maintenance of the "steady state." This constancy is brought about by adequate composition and transport of a system of body fluids.

The human body is made up of about 70 per cent water by weight. About one half, or 50 per cent, of the weight is water within the cell (intracellular fluid, ICF). The remainder of the water, amounting to 20 per cent of body weight (see Fig. 9) is outside the cell (extracellular, ECF). The extracellular water is distributed in two fluid compartments: the vascular compartment, or plasma, which amounts to 5 per cent of body weight; and the intercellular (or interstitial) space, which amounts to 15 per cent of body weight. According to these figures, the average (70 kg.) man would have about 49 L. of total body water: 50 per cent, or 35 L., as ICF, and 20 per cent, or 14 L., as ECF. About 15 per cent, or 10.5 L., would be intercellular, and about 5 per cent, or 3.5 L., would be plasma.

The fluid compartments of the body are separated from each other by membranes through which interchange takes place constantly. This interchange is determined by reactions of a physical, chemical and biologic nature. Changes in one type of body fluid result in changes in other fluids. Alterations in the fluid of the vascular compartment affect both the fluid of the intercellular compartment and the fluid of the intracellular compartment. These alterations concern the volume, tonicity and chemical composition, and acid-base balance of the body fluids.

The capillary membrane is relatively impermeable to the passage of plasma proteins but permits passage of many other constituents. The rate of fluid exchange at the capillary wall is determined by a balance between capillary blood pressure opposed by the colloid osmotic pressure (plasma proteins) plus the tension in the tissues. At the arterial end of the capillary, where blood pressure is high compared

CHAPTER 4

Fluid and Electrolyte Balance

IT SHOULD BE emphasized at the outset that the great majority of problems in the area of fluid and electrolyte balance are not beyond the ability of the physician to analyze and resolve. In order to do so, however, he must possess a basic understanding of normal water and electrolyte balance, the factors which lead to disturbed relationships, the normal compensatory mechanisms which operate, the means by which abnormal states may be recognized and the measures available for their correction

One may develop a concept of these problems from a chapter such as this, but real understanding and ability to apply the available knowledge at the bedside comes only with sustained intensive study, including wide reading, many discussions, repeated observations and actual practice. There are neither short cuts to this knowledge nor ready-made descriptions which apply in all situations. The over-all treatment of the patient must be the primary consideration, and management of an existing fluid and electrolyte problem is but one facet The patient who has suffered serious depletion may die despite the fact that his body fluids have been restored to normal. Likewise, undertreatment, as well as overtreatment, can be fatal.

Claude Bernard in 1878 pointed out the importance of regulatory mechanisms in the preservation of the constancy of the internal environment or *milieu intérieur*. This constancy of the internal environment is maintained largely by the nature of the medium in which the cells function. The medium and the cells are contained by the skin, a relatively impervious membrane surrounding the entire body. The alimentary tract provides a mechanism for replenishing the medium;

Chemistry of the Body Fluids

In the ECF, the principal cation is sodium. The anions are chloride and bicarbonate, with smaller amounts of phosphate and sulphate.

Within the cell, potassium is the principal cation. Water is freely transferable between the ICF and the ECF. The concentration of sodium and potassium on each side of the cell membrane varies according to changes in osmotic pressure and cellular metabolism. In general, the osmotic pressures of the ICF and ECF are equal.

When osmotic equilibrium is disturbed from loss of the principal extracellular cation (sodium) or from retention of water, the ECF osmotic pressure falls, and a prompt shift of water into the ICF results. This produces a rise in ECF osmotic pressure and a fall in ICF osmotic pressure until balance is restored.

The application of chemical equivalents to the measurement of electrolytes is necessary for a clear understanding of these problems. The student should review this subject in his textbook of biochemistry. Only a few aspects can be considered here.

If the weight in grams of NaOH, KOH, and HCl, corresponding to the molecular weight of these substances, is each dissolved in 1 L. of water, the result is three molar solutions, each with 1 mole of the dissolved substance per liter. The solutions will contain 40 Gm. of NaOH, 56 Gm. of KOH and 37 Gm. of HCl. Equal volumes of the alkaline solutions will neutralize equal volumes of the acid solution. The NaOH solution and the KOH solution, therefore, may be said to have the same combining power and the same number of active particles (cations and anions) per unit volume. These solutions are *equivalent*, mole for mole. The substances dissociate into anions and cations; each solution, therefore, contains one equivalent of anion and one equivalent of cation.

Substances react on the basis of their valence. The above-mentioned substances all have a valence of one. Calcium, on the other hand, is a bivalent atom with an atomic weight of 40. One mole of calcium (40 Gm. dissolved in 1 L. of water) possesses twice the combining power of 1 mole of sodium. Therefore, 1 mole of calcium is two equivalents of calcium, and one equivalent weight of calcium weighs 20 Gm.

Because the concentration of electrolytes in the blood is low and because it is easier to express their values in whole numbers, the unit of measure used is 1/1,000 of an equivalent, or milliequivalent (mEq.).

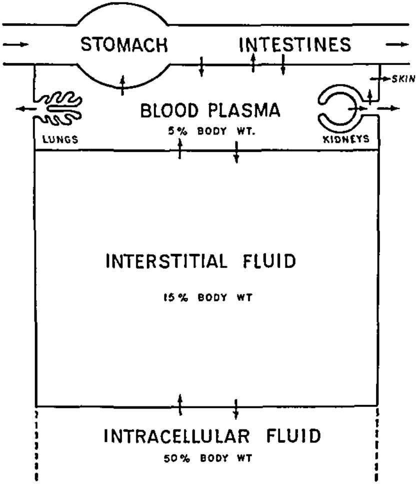

Fig. 9—The distribution and interchange of body water. The blood plasma (5 per cent of body weight) and the interstitial fluid (15 per cent) constitute the extracellular fluid (ECF), which comprises about 20 per cent of the total body weight (From Gamble, J L *Chemical Anatomy, Physiology and Pathology of the Extracellular Fluid· A Lecture Syllabus* [Cambridge, Mass., Harvard University Press, 1950])

with osmotic pressure, an ultrafiltrate of the plasma leaves the capillary and enters the tissues. At the venous end of the capillary, where blood pressure is low compared with the osmotic pressure, resorption of tissue fluid occurs (Starling's hypothesis).

Dissolved solutes tend to move in the direction of exuded, or resorbed, fluid, but they also diffuse from regions of higher to lower chemical concentration. This may lead to the transfer of an electrolyte against the direction of fluid flow.

the H^+ ion concentration drops and the solution becomes more alkaline. If, on the other hand, more H^+ ions are added in the form of carbonic acid ($HHCO_3$), the pH is lowered and the solution becomes more acid. The concentration of the common ion (HCO_3), then, determines the reaction of the solution.

The concentration of bicarbonate in the plasma is dependent on the amount of base (cations) available to form bicarbonate at the *particular CO_2 tension* with the particular amounts of blood electrolytes. The base available to form bicarbonate is regulated by the kidneys. Increases or decreases in the total cations, by increasing or decreasing the bicarbonate, cause variation in the plasma pH by the common ion effect. Most of the fixed cations of the blood are combined with chloride and other fixed anions, except for that which is combined with bicarbonate (27 mEq./L.) and protein anion. The measure of base available for combination with CO_2 is the CO_2-combining power. The CO_2-combining power represents the quantity of CO_2 present as bicarbonate in the plasma ($BHCO_3$).

The concentration of carbonic acid is variable according to pulmonary ventilation and is normally stabilized at 1.35 mEq./L.

The normal pH of the blood (7.4) is maintained when the ratio of $BHCO_3$ to $HHCO_3$ is $\frac{27 \text{ mEq./L.}}{1.35 \text{ mEq./L.}}$, or *20:1 according to the* Henderson-Hasselbalch equation,

$$pH = 6.1 + \log \frac{BHCO_3}{HHCO_3}$$

Although the total amount of bicarbonate and carbonic acid may vary within a wide range, so long as the relationship of 20:1 remains stationary, the pH of the blood is unchanged. The derivation and application of the Henderson-Hasselbalch equation to acid-base balance should be reviewed.

The rapidity with which the system of bicarbonate buffers acts through the respiratory mechanism makes it an important stabilizing influence in body economy.

Intake and Output

Water is taken into the body (Fig. 10) either by direct ingestion through the gastrointestinal tract or as a result of water released by oxidation of foods. Although normal intake of water varies greatly according to age, weight, environmental temperature, disease, fever

Under normal conditions, the plasma contains approximately 155 mEq./L of cations and an equal number of anions. All the components except protein are readily diffusible into the interstitial fluid. *The ionic concentration of interstitial fluid is, therefore, the same as* that in the plasma. If the same unit of measurement (mEq.) is used and total concentration of cations and anions is determined, the total concentration of cations is always equal to that of the anions. The student should memorize the values of the usually measured plasma electrolytes. They are: Na 142, K 5, Cl 103 and CO_2 27 mEq./L.

It is possible to convert values expressed as milligrams per 100 ml. to milliequivalents per liter by use of the formula:

$$\frac{\text{Mg}/100\text{ ml.} \times 10}{\text{Atomic weight} \times \text{valence}} = \text{mEq./L.}$$

Changes in acid-base balance may be defined as deviations from normal reaction, or pH of the blood. The pH of the blood is 7.4, and normal variations between 7.35 and 7.45 are noted. Acidosis is that state in which the forces are acting to increase the hydrogen-ion concentration (decrease the pH), and alkalosis is that state in which the forces are acting to decrease the hydrogen-ion concentration (increase the pH) Several compensatory mechanisms operate to maintain the narrow limits of the reaction of the blood and to prevent the development of acidosis or alkalosis. The major role is played by the plasma buffers (bicarbonate, phosphate and protein), the excretion of CO_2 by the lungs and the excretion of fixed acids by the kidneys.

The pH of the blood is determined by the ratio of carbonic acid to bicarbonate of the plasma Carbon dioxide is available in large amounts as an end product of metabolism. It is picked up by the blood and transported to the lungs for excretion. The concentration of carbonic acid of the plasma depends on the partial pressure of CO_2 (pCO_2) in the arterial blood, which is equilibrated with the partial pressure of CO_2 in the lung. If the metabolic rate is constant, the pCO_2 of the lung and plasma depends on changes in pulmonary ventilation, which are subject to regulation by the respiratory center. Thus, when alveolar pCO_2 is increased (hypoventilation), the plasma pCO_2 increases, and when alveolar pCO_2 is decreased (hyperventilation), the plasma pCO_2 decreases.

The law of the "common ion effect" is involved in acid-base balance. If to a solution of carbonic acid ($HHCO_3$) which is weakly ionized, HCO_3 in the form of sodium bicarbonate ($NaHCO_3$) is added,

through the stool, the lungs and the skin is relatively constant. Water loss through the kidneys fluctuates markedly, depending on the amount of water available for urinary excretion after other demands for water have been met. Generally, the urinary output will range from 1,000 to 1,800 ml./day under normal conditions.

Water loss by evaporation through the skin and lungs constitutes an important mechanism for the preservation of a constant body temperature. Loss through these routes proceeds without regard to

TABLE 2.—Approximate Daily Loss and Requirement of Water for Patients *

Age Group	Water Loss (Ml./Day)				Usual Allowance (Ml./Kg.)
	Urine	Stool	Insensible	Total	
Infants	200–500	25–40	75–300	300–600	125
Children	500–800	40–100	300–600	800–1,500	75
Adults	700–1,000	100	600–1,000	1,500–2,000	45

* Note that the values for healthy, vigorous adults given in the text are greater.

the amount of water present in the body stores. These routes of water loss, therefore, may be said to have *preferential* rights to available body water.

The kidneys of the normal healthy adult must remove 35 Gm. of solid material, or waste matter, from the blood each day. Unless this is accomplished, these products will accumulate in the blood and the state of equilibrium or health is upset. When kidney function is impaired and the kidneys are unable to concentrate the urine, the volume of water needed for excretion of metabolic waste products is increased. If there is insufficient water for a large output of dilute urine, waste products will tend to accumulate in the body. On the other hand, when the kidney is able to concentrate the urine through tubular reabsorption of water, a relatively small volume of urine will suffice to carry away the necessary amount of waste products. Thus, when the specific gravity of the urine approximates 1.030, only about 500 ml. of urine is needed to remove 35 Gm. of waste products, whereas with a low specific gravity of 1.010, about 1,500 ml. or more of urinary excretion is needed to accomplish the same result. Thus, in health, sufficient water intake consists of that amount which is regularly lost through

and many other factors, the intake of a healthy adult approximates 2,000–2,500 ml./day. This is usually supplied by about 1,200 ml. from liquids taken orally, 1,000 ml. from water of solid foods and 300 ml. from water of oxidation. These values do not apply in infants and children, where the turnover of water is relatively much greater.

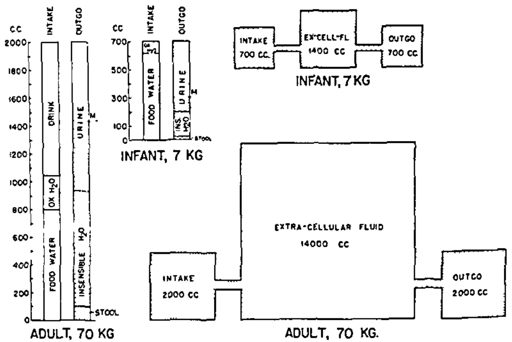

FIG 10.—Water intake and output in the adult and infant. *M*, minimal water expenditure/day (about 300 ml. in the infant and 1,400 ml in the adult). *To left,* relation of body weight to water exchange. Although the infant weighs only 1/10 as mu
relatic
ECF
daily
ECF in the adult (From Gamble, J. L.: *Chemical Anatomy, Physiology and Pathology of the Extracellular Fluid· A Lecture Syllabus* [Cambridge, Mass.: Harvard University Press, 1950])

Water is lost from the body through several routes (Fig. 10 and Table 2). The least important is the loss in the stool, which amounts to only about 100 ml./day. Water loss in the expired air is about 500 ml., and water loss from the skin by evaporation (insensible perspiration) is also about 500 ml./day. When perspiration is increased as a result of an increase in environmental temperature, the loss may be far in excess of this amount, and significant degrees of fluid and electrolyte depletion may result. In health, the loss of water

also exemplified by kidney function. In the average person, the glomerular capillaries filter out approximately 180 L. of extracellular-like fluid daily, but 99 per cent of the water and more than 99 per cent of the sodium chloride are reabsorbed in the renal tubules and retained for body economy.

SALT AND WATER

The substances dissolved in the ECF determine its osmotic pressure The electrolytes, sodium and chloride, are in highest concentration, and nonelectrolyte substances, such as glucose and amino acids, are in lower concentration. Sodium plays the most important role in determining the volume and tonicity of the extracellular fluid. When sodium is lost, osmotic tension falls and water leaves the extracellular space; and, conversely, when sodium is retained, water enters the ECF until osmotic equilibrium of all body fluids is restored.

The sodium content of the ECF is under the control of kidney function. The concentration of sodium in the plasma determines the level of sodium resorption in the kidney tubules. When the plasma sodium concentration is greater than normal, the antidiuretic hormone (ADH) released from the posterior pituitary causes increased water absorption in the tubules, and the concentration of sodium in the blood is lowered. On the other hand, when the plasma sodium concentration is lower than normal, hormones of the adrenal cortex (DCA-like mineral corticoids) act upon the tubules to increase sodium resorption and thus increase the plasma sodium concentration. This brief description of the hormonal factors concerned in renal regulation of salt and water balance is a much oversimplified version of the complex changes involved.

Water and salt balance may be considered adequate, provided renal and adrenal function is normal, if the daily urine volume exceeds 1,000 ml. and if the salt concentration of the urine approximates 3 Gm./L. The urinary salt excretion can be estimated simply by use of the Fantus test.* Although the urinary output of salt is not neces-

* The Fantus test is performed on a sample of 10 drops of urine. Potassium chromate is used as the indicator. Silver nitrate (2.9 per cent solution) is added, drop by drop, until a permanent and distinct change in color (from yellow to red-brown) takes place. Provided the same dropper is used to measure both the silver nitrate solution and the urine, and the dropper is rinsed in distilled water after the urine sample is measured, the number of drops required to cause the color change will be approximately equal to the number of grams of sodium chloride present per liter of urine.

the skin and lungs plus the amount needed for urinary excretion, or 2,000–2,500 ml. every twenty-four hours.

Just as there is need for replenishment of water, there is need for replenishment of electrolytes. A healthy adult living in a temperate climate will need about 5 Gm. of sodium chloride a day. Amounts in

TABLE 3.—TOTAL VOLUME OF DIGESTIVE SECRETIONS PRODUCED IN 24 HOURS BY ADULT OF AVERAGE SIZE*

Saliva	1,500 cc.
Gastric secretions	2,500 cc.
Bile	500 cc.
Pancreatic juice	700 cc.
Secretions of intestinal mucosa	3,000 cc.
	8,200 cc
Blood plasma volume	3,500 cc.

* From Gamble, J L *Chemical Anatomy, Physiology and Pathology of Extracellular Water* (Cambridge, Mass : Harvard University Press, 1950)

TABLE 4.—ELECTROLYTE COMPOSITION OF THE INTESTINAL FLUIDS (AVERAGE FIGURES)*

SOURCE	Na^+ IN mEq./L.	K^+ IN mEq./L.	Cl^- IN mEq./L.
Gastric	60	9	84
Small bowel (Miller-Abbott tube)	111	5	104
Ileostomy			
Recent	130	11	116
Adapted	46	3	21
Proximal colostomy or cecostomy	52	8	42
Biliary drainage	130	5	100
Pancreatic drainage	140	5	77
Transudates	130–145	3–5	90–100

* From Lockwood, J S, and Randall, H. T Bull New York Acad Med 25 228, 1949.

excess are readily excreted by the kidney. Generally, the sodium chloride, potassium, and small amounts of calcium and magnesium required to maintain normal metabolism are well covered by an adequate dietary intake.

The body conserves water and electrolytes to an amazing degree. For example, each day a great quantity of water and electrolytes, amounting to 8 or 10 L. of extracellular-like fluid, is secreted into the gastrointestinal tract (Tables 3 and 4), where nearly all is reabsorbed and used over and over. This conservation on the part of the body is

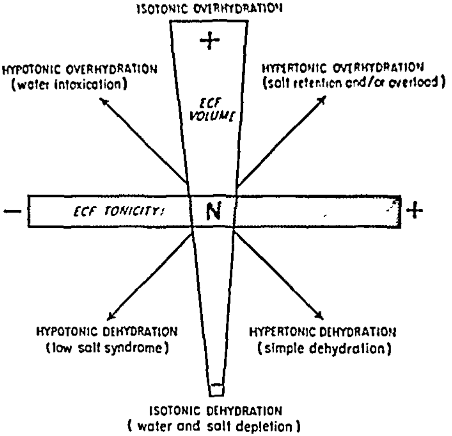

FIG. 11.—*Diagrammatic representation of the directions in which disturbances of* ECF volume and tonicity may proceed. The area marked *N* indicates normal ECF volume and tonicity. The vertical bar indicates volume, changes of which are suggested by changes in width of the bar. The horizontal bar indicates tonicity (osmolarity), changes of which are indicated by changes in the number of dots (ions). The possible variations in volume and tonicity are indicated by arrows. Dehydration states lie below the horizontal bar, overhydration states above the horizontal bar. Management of each type of disturbance can be based on an understanding of these relationships.

central nervous system depression with weakness, apathy, anorexia and sleepiness. When the volume deficit is great, stupor and coma may be observed. The reduced fluid volume produces alterations in blood volume and blood flow. Tissue changes include furrowing and dryness of the tongue, loss of subcutaneous turgor, soft eyeballs and atonic muscles. Disturbances in gastrointestinal function, such as nausea and vomiting, may also appear.

Correction of this deficiency requires the administration of repair solutions equal in volume and composition to that which has been lost from the ECF.

sarily an accurate index of electrolyte disturbances, the test nevertheless provides a practical method for estimating renal retention or loss of salt and the patient's need for more or less salt.

In normal persons who have taken large amounts of water, chloride may be absent from the urine. If the urine is concentrated and the chloride content is less than 3 Gm./L., salt depletion must be considered. If the urine contains more than 5 Gm./L. and there is no evidence of renal or adrenal insufficiency, it is unlikely that the patient is suffering from salt depletion. This test must be interpreted in terms of other observations and correlated with them.

Dehydration States

Disturbances in water and electrolyte balance develop as a result of (1) inadequate or excessive intake, (2) renal or extrarenal losses and (3) distributional shifts such as exudation into the body cavities or into tissues (e.g., peritonitis, burns, fractures). In surgical cases, losses through the gastrointestinal tract and distributional shifts account for most imbalances, and overloads are a less common cause of imbalances. Renal failure may also complicate the picture.

According to the circumstances, the loss of water and electrolytes may proceed at relatively the same rate, water loss may exceed electrolyte loss, or electrolyte loss may exceed water loss. The alterations in the fluid volume and tonicity which result under these conditions may be termed "isotonic dehydration," "hypertonic dehydration" and "hypotonic dehydration," respectively. The recognition of dehydration states according to this classification simplifies their management. Deficiencies of these types are most often encountered by the clinician (Fig. 11)

Isotonic Dehydration (Salt and Water Deficit)

Isotonic dehydration results from the loss of ECF. It may be due to vomiting; diarrhea; intestinal, pancreatic or biliary fistulas, suction drainage of the small bowel, ileostomy drainage; burns; excessive sweating without fluid replacement; etc. The chemical composition and tonicity of the plasma remains within relatively normal limits, but the volume of the ECF is decreased.

The diagnosis of isotonic dehydration is based upon an evaluation of the history, physical findings and intake-output data. The blood chemistry studies may reveal no significant abnormalities. There is

If possible, the oral route is used, otherwise, 5 per cent glucose solution is given parenterally. Because water loss almost never occurs without some salt loss, both water and salt are required for repair.

Hypotonic Dehydration (Salt Loss with Slight Water Loss; "Low Salt Syndrome"; "Water Intoxication")

Hypotonic dehydration (Table 6) may result from extrarenal and renal salt losses (e.g., "salt-losing syndrome," Addison's disease) when

TABLE 6 – Grades of Salt Depletion *

Grade	Signs	Salt Deficit	Approximate Deficit of Isotonic Saline in a 70 Kg. Man
I Slight to *moderate*	May be lassitude, giddiness, *orthostatic fainting*. Chloride in urine low or absent	Up to 0.5 Gm./kg body weight	Up to 4 L.
II. Moderate to severe	Lassitude, giddiness and fainting, anorexia and *may be nausea and vomiting*. Fall in blood pressure. Chloride in urine absent (except in Addison's disease)	0.5–0.75 Gm./kg. body *weight*	4–6 L.
III Severe to very severe	Apathy, stupor, vomiting, and systolic blood pressure less than 90 mm Hg. Chloride absent in urine (except in Addison's disease)	0.75–1.25 Gm./kg. body weight	6–10 L.

* From Marriott, H L. *Water and Salt Depletion* (Springfield, Ill., Charles C Thomas, Publisher, 1950).

water is replaced without salt. Under these conditions, the water loss is replaced in part by oral or parenteral intake but the electrolyte loss is absolute. This occurs, for example, when a patient who is losing large amounts of ECF through diarrhea continues to drink water without salt. *Electrolyte depletion with hypotonicity of the body fluids results* Hypotonicity of the ECF will lead to transfer of fluid into the ICF, and a state of water intoxication develops.

The renal mechanism for conservation of salt will be invoked; but

Hypertonic Dehydration (Water Loss with Slight Salt Loss)

Hypertonic dehydration (Table 5) may be caused simply by the inability to drink water (e.g., esophageal obstruction). This type of dehydration usually develops slowly. There is continued expenditure of water for metabolic needs and an inadequate water intake, which results in decreased volume and hypertonicity of the ECF. Fluid then

TABLE 5 – Grades of Water Depletion *

Grade	Signs	Body Weight Deficit	Approximate Deficit in a 70 Kg Man
I Early	Thirst, other effects not yet present	2%	1.5 L
II Moderately severe	Marked thirst and dryness of mouth, oliguria, weakness, ill appearance, *slight personality* changes, still capable of fair mental and physical performance	6%	4.2 L.
III Very severe	All the above manifestations with in addition marked impairment of mental and physical capacity	7–14%	5–10 L

* From Marriott, H. L. *Water and Salt Depletion* (Springfield, Ill.: Charles C Thomas, Publisher, 1950)

shifts from the intracellular into the extracellular space, producing true cellular dehydration.

The mouth and mucous membranes become dry. The skin may be flushed and hot, and body temperature is elevated. Weakness, weight loss and psychic changes may be noted.

The output of urine is decreased, and its specific gravity is high. The urine often contains albumin, red cells and casts, and the chloride concentration of the urine is increased. The red cell count and hematocrit are both elevated. The plasma electrolyte concentration and tonicity of the ECF is increased.

The treatment of hypertonic dehydration consists in giving water.

slowly) may be administered. If the disturbance is less severe, isotonic saline and M/6 sodium lactate, in the ratio of 4 to 1, is given. If there has been little salt loss with water overloading, simply withholding water and administering salt (orally) may correct the imbalance.

Potassium Balance

In health, the potassium intake is maintained by diet and averages 70–100 mEq. (2.8–4.0 Gm.) daily. Potassium is most abundant in the cell, where its concentration is about 25 times greater than in the ECF. Excretion of potassium occurs, for the most part, through the kidney. The kidney is not able to conserve potassium as it does sodium, and under conditions of normal renal function, excesses of potassium are rapidly excreted. When there is impaired renal function (oliguria, anuria), potassium tends to accumulate in the ECF and may lead to potassium intoxication.

Hypopotassemia (Hypokalemia)

Hypopotassemia, rather than hyperpotassemia, is more commonly encountered in surgical practice. The recognition of both states requires an alertness to the conditions which lead to them and a knowledge of the clinical manifestations which each produces. It must be borne in mind that the plasma K^+ concentration, as indicated by flame-photometric determination, does not necessarily reflect the K^+ concentration of the ICF.

Potassium deficits develop under a variety of conditions:

1. Starvation from prolonged parenteral fluid therapy without potassium replacement.
2. Severe dehydration states.
3. Alkalosis due to predominant chloride loss from vomiting, diarrhea, suction drainage, fistulas, etc. The resulting hypochloremic alkalosis can be corrected by parenteral therapy only if the potassium deficit is relieved.
4. Hypopotassemia, which may follow correction of acidosis without potassium replacement. This occurs during the recovery phase of diabetic acidosis when potassium is not supplied.
5. Following traumatic shock due to crush injury, thermal burns or injuries due to cold, when potassium diuresis may be excessive.

in spite of this, if there is continued salt loss without replacement, the concentration of electrolytes falls.

The clinical signs are related to hypotonicity of all body water and decreased volume of the ECF. There is marked muscle weakness, often with cramps and twitching. Striking central nervous system changes, such as stupor, confusion, coma, convulsions, psychotic behavior and "strokes," may appear. Gastrointestinal disturbances, including anorexia, nausea, vomiting, inhibition ileus and false signs of peritonitis, are common.

TABLE 7 – COMPARISON OF EFFECTS ON WATER AND SALT DEPLETION*

MANIFESTATION	PURE WATER DEPLETION	PURE SALT DEPLETION
Dehydration	+++ primary or simple	+++ secondary or extracellular
Thirst	+++	Absent
Lassitude	+	+++
Orthostatic fainting	Absent until late	+++
Urine volume	Scanty	Normal until late
NaCl in urine	Often +	Always absent except in Addison's disease
Vomiting	Absent	May be +++
Cramps	Absent	May be +++
Plasma NaCl	Slight increase or normal	Diminished +++
Blood urea	+	+++
Plasma volume	Normal until late	Decreased +++
Hemoconcentration	Normal until late and slight	+++
Blood viscosity	Normal until late	Increased +++
Blood pressure	Normal until late	Fall +++
Water absorption	Rapid	Slow
Mode of death	? due to rise of osmotic pressure	Peripheral circulatory failure

* From Marriott, H. L. *Water and Salt Depletion* (Springfield, Ill.: Charles C Thomas, Publisher, 1950)

As the volume of the ECF decreases, peripheral vascular failure develops. There is tachycardia, hypotension, a tendency to fainting in the upright position (orthostatic syncope), decreased cardiac output and, ultimately, signs of cardiac failure. With fall in renal blood flow, retention of metabolites and uremia develops. The urine output decreases, specific gravity is low, and chloride output is negligible. The concentration of the plasma electrolytes is decreased.

Treatment will depend on the severity of the volume and osmotic deficits (Table 7). Signs of diminished circulating blood volume call for blood transfusions. If there is a marked decrease in ECF tonicity, small volumes of hypertonic salt solution (200 ml. of 3 per cent NaCl,

Volume and ionic deficits must be repaired. Infusion of a glucose-insulin solution is recommended. In most cases of acute renal failure, the three-phase management, described elsewhere, will tide the patient over until diuresis occurs. If such management appears unlikely to succeed, one must consider external dialysis (artificial kidney) or some means of internal dialysis. (See the discussion on acute renal failure on page 294.)

DATA GATHERING

HISTORY.—The history of the illness should include the patient's normal weight, weight loss or gain, food and fluid intake, and normal and abnormal output. This information will often indicate the presence of a fluid-balance problem, its origin and its relative magnitude.

Symptoms which result from fluid and electrolyte disturbances are variable, nonspecific and often overlooked. Salt depletion (water intoxication) may be associated with lassitude, anorexia, asthenia, nausea and vomiting, blurred vision, muscle cramps, convulsions and coma. Thirst is prominent with water loss and hypertonicity of the body fluids. Acid-base imbalance is usually associated with changes in rate and depth of pulmonary ventilation. Hypopotassemia may be suggested by apathy, weakness, drowsiness and muscle paralysis.

PHYSICAL FINDINGS.—Ventilatory overactivity or underactivity may suggest metabolic acidosis or alkalosis, respectively. The possibility of primary respiratory abnormalities producing disturbed ventilation must, of course, be considered. Furthermore, one should keep in mind that, when hydrogen-ion concentration is seriously disturbed, the activity of the respiratory center is inhibited. Thus, at moderate concentration, CO_2 is a powerful stimulant to ventilation, but at high levels it is a depressant. The signs and symptoms must, therefore, be interpreted in light of all other findings. To dwell too much on one sign, or one laboratory test, to the exclusion of the entire clinical picture, will lead to distortion and failure to analyze the patient's condition correctly.

The head should be examined carefully. In the infant, depression of the fontanelles and eyes, and in the adult, softening of the eyeballs, dry mouth and furrowed tongue signify a depletion of the ECF volume. Changes in elasticity, moisture, temperature and turgor of the skin are also important signs. The differences in elasticity of the skin that are normal in youth and old age should be learned by observa-

The clinical signs of potassium deficiency include: striking muscle weakness, transitory periods of muscle paralysis, dyspnea with gasping respirations, anorexia, nausea, vomiting, distention, "chronic ileus" and cardiac failure with characteristic electrocardiographic changes. The triad of hypochloremia-alkalosis-acid urine is usually indicative of a potassium deficit. The serum potassium level may be lower than 4 mEq /L.

The electrocardiographic abnormalities include: lengthening of the Q-T interval, depression of the S-T segment and depression or inversion of the T wave.

Potassium salts can be given orally, subcutaneously or intravenously, but the oral route is preferred whenever available. When kidney function is normal and hydration adequate, oral administration will rarely produce potassium intoxication. Potassium chloride tablets may be used and can be given in amounts of 5–10 Gm./day in divided doses.

Parenteral potassium therapy may be supplied in the form of Darrow's solution or ampules of KCl containing 30 or 40 mEq. of K^+ added to 1 L. of any type of repair solution—electrolyte, glucose or amino acid. An intake of 40 mEq. of potassium daily is adequate for metabolic needs. Repair of deficits requires about 30–40 mEq. (1 ampule) for each liter of electrolyte solution required. It must be reemphasized that potassium salts must be given slowly (not over 20 mEq /hr), and only when urinary output is satisfactory.

Hyperpotassemia (Hyperkalemia)

Elevated serum potassium levels and potassium intoxication are infrequent clinical findings, but they may occur with impaired renal function (oliguria or anuria), adrenocortical failure (Addison's disease), early diabetic acidosis before treatment is started and parenteral potassium overdosage.

The most important clinical manifestations are the result of the toxic effect of potassium on the function of the heart Irregularities in cardiac rate and rhythm, peripheral vascular collapse and cardiac arrest occur. The serial electrocardiographic changes are specific and can be correlated with increases in the serum potassium. Cardiac arrest and death usually occur when the level reaches 10–11 mEq./L.

Treatment of hyperpotassemia consists in discontinuing potassium if it is being administered and, if possible, increasing urinary excretion

Volume and ionic deficits must be repaired. Infusion of a glucose-insulin solution is recommended. In most cases of acute renal failure, the three-phase management, described elsewhere, will tide the patient over until diuresis occurs. If such management appears unlikely to succeed, one must consider external dialysis (artificial kidney) or some means of internal dialysis. (See the discussion on acute renal failure on page 291.)

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Treatment of hyperpotassemia consists in discontinuing potassium if it is being administered and, if possible, increasing urinary excretion.

Simple Laboratory Aids.—Additional information may be derived from simple bedside laboratory tests of the urine specific gravity and pH, urinary chloride, blood hemoglobin, red and white cell counts and hematocrit. The pH of suction fluid (and other fluids) can be determined by use of nitrazine paper. Urine sodium chloride output can be estimated by the Fantus test.

The application of simple diagnostic methods may be illustrated by the following observations. A patient who has been vomiting highly acid gastric juice (as indicated by testing it with nitrazine paper) is also breathing slowly and deeply. The presumption is that he has metabolic alkalosis which is partially compensated by decreased pulmonary ventilation. The urine reaction is highly alkaline, and urine sodium chloride is low or absent. These findings substantiate the diagnosis of metabolic alkalosis.

Blood Chemistry Determination.—If available, more complicated laboratory tests, including determinations of the serum Na, K, Cl and CO_2-combining power, are often desirable. Serial electrocardiographic tracings are sometimes indicated to detect changes in potassium levels as reflected by alterations in myocardial conduction.

Blood chemistry determinations are helpful in establishing the tonicity (osmolarity), chemical composition and acid-base relationships of the ECF. Disturbances in these areas can often be suspected on the basis of the clinical examination, and certainly these tests are not always necessary.

The concentration of cations and anions in the ECF is always equal, and normally amounts to 155 mEq./L. of each. Sodium is the most important ECF cation; and if it is present in a concentration ranging from 138 to 142 mEq./L., the ECF tonicity (osmolarity) is probably normal.

The anions of the ECF include Cl^- and $BHCO_3^-$, which are often measured, and sulfates, phosphates and organic acids, which are usually not measured. The $BHCO_3$ is determined by measuring the CO_2-combining power. The sum of these anions (in mEq./L.) normally approximates the sodium concentration. Thus, Cl (103) plus CO_2-combining power (27) plus other anions (about 12) equals 142 mEq./L. From a practical standpoint, if the sum of Cl and CO_2-combining power is within the range of 125–135 mEq./L., and the patient exhibits no cyanosis, acetonuria or other signs of metabolic imbalance, the tonicity of the ECF is probably normal.

The CO_2-combining power must be interpreted in the light of

tion in order better to evaluate alterations in dehydration states. The lung fields should be examined for signs of pulmonary congestion, which may be due to excessive ECF, heart failure, inadequate colloid osmotic pressure of the plasma, increased production or decreased destruction of the antidiuretic hormone, altered adrenocortical output or a combination of factors. In the differential diagnosis of pulmonary edema, one must consider the entire mass of information: cardiac status, venous pressure, peripheral edema, ascites, liver size and tenderness, etc

Much information can be gained from noting the tone, fulness and strength of the muscles. Potassium deficiency (hypopotassemia) from loss of ICF usually produces alterations in muscle function. There may be interference with neuromuscular activity and a decrease in reflex responses as a result of changes in nerve conduction. Abnormalities in myocardial conduction, generalized muscular weakness, and even respiratory paralysis, may develop. Hyperpotassemia is uncommon in surgical patients except when it is induced therapeutically, in which case cardiac irregularities, including cardiac arrest, may appear. Abnormalities in calcium metabolism also affect neuromuscular activity. Hypocalcemic tetany often follows surgical removal of the parathyroid glands. Tetany may also develop in severe alkalosis or during the recovery phase of rickets. Accompanying or preceding overt tetany, evidence of neuromuscular hyperirritability (Chvostek's sign, Trousseau's sign, carpopedal spasm, increased tendon reflexes) appears.

Faintness and orthostatic hypotension may result from inadequate ECF volume, simple low blood volume, lowered blood osmotic pressure, "chronic shock" of nutritional depletion or primary cardiac disease. In some cases an "exercise test" is necessary to demonstrate such abnormalities

If volume deficiency is severe, tachycardia and hypotension may be noted. In acute depletion states, the signs are often striking; but in the slowly developing hypovolemia of malnutrition, special observations are sometimes required to bring out circulatory inadequacies.

Intake and Output Record.—An important part of the data required for analysis of these problems is provided by an accurate intake-output record. Previous losses must be estimated, current losses calculated and future losses projected, in order that water and salt needs can be repaired and maintained. These records must include the quantity and composition of the fluids involved

renal insufficiency or an existing fluid deficiency. If the infusion results in prompt diuresis, a state of dehydration and the need for water is established. Such attempts at diagnosis by noting the response to treatment must be carefully controlled; and if the suspected diagnosis is not supported by an appropriate response, therapy must be terminated before damage occurs.

Once mastered, basic principles may be applied to each case in a specific, systematic and rational manner. Because no two cases are exactly alike, it would be misleading to attempt to discuss all types of derangements. Specific problems involving fluid and electrolyte disturbances of infants and children, or of patients with diabetes mellitus, cardiac failure, central nervous system disorders or renal and adrenal dysfunction, may require specific treatment. While treatment *in each specialty and each disease state may vary, the broad general* principles of therapy are the same.

Special Problems in Infants and Children

Therapy in infants and children requires that the physician be acutely aware of the relative magnitude of fluid volume and rate of turnover. In early life, the ECF may represent up to 40 per cent of the body weight. Glomerular filtration per square meter of body surface is only about 50 per cent of that of the adult, and tubular function is less capable of adjustment to metabolic and therapeutic errors. The younger the patient, the greater is the danger of giving too much fluid and salt. Fluid balance problems in premature infants may require hourly supervision. Probably more infants succumb to overhydration than underhydration. The temptation to give a little extra fluid as long as an infusion is working must be resisted.

The daily maintenance requirements are between 40 and 125 ml./kg. of body weight. The small infant requires the largest amount per kilogram. Five or 10 per cent glucose is administered for fluid maintenance. Electrolytes are given for repair of deficiencies, such as when gastric juice or other electrolyte-containing solutions have been lost. They should be replaced in matching quantity and kind. Blood and blood substitutes should not be given until the deficiency in blood volume has been actually calculated. In general, a single transfusion should not exceed a volume of 25 ml./kg. The usual volume is 15–20 ml./kg

the clinical findings and, if available, the blood pH. The CO_2-combining power is decreased in metabolic acidosis and respiratory alkalosis. It is increased in metabolic alkalosis and respiratory acidosis. When secondary changes have occurred through the buffer systems of the blood and through respiratory and renal mechanisms, the blood pH may be normal with either a decreased or increased CO_2-combining power value The physician must remember that the CO_2-combining power may be either high or low in either acidosis or alkalosis.

The results of all tests must be interpreted in terms of all other available information. It is a common error to confuse abnormalities in tonicity with those of chemical composition. If plasma sodium is high (or low), then all other ions must be proportionately high (or low) if tonicity of the extracellular fluid is actually altered. A deviation in the CO_2-combining power means nothing with regard to acid-base balance unless the deviation is dissimilar to, or out of proportion to, deviation in the sodium concentration. For example: alterations in CO_2-combining power may result either from abnormalities in acid-base balance or from abnormalities in ECF tonicity. A normal CO_2-combining power associated with hypotonicity of the ECF may indicate an existing metabolic alkalosis, while a normal CO_2-combining power associated with hypertonicity may indicate an existing metabolic acidosis. For these reasons, the entire electrolyte picture must be evaluated if a complete diagnosis is desired (Fig. 14, p. 100).

It is also a common error to forget that a considerable piling-up of other anions (sulfates, phosphates and ketone bodies) may occur, since they are not usually measured. Thus, if the physician depends upon laboratory values without circumspection, he may be hindered rather than helped. Whenever a single laboratory value is out of line with all the other evidence, it should be repeated or discounted. Treat the patient, not a lab report.

WATCHING THE EFFECT OF TREATMENT.—Lastly, the response to treatment *(diagnosis ex juvantibus)* is a helpful, but often neglected, aid to diagnosis. For example: A patient who is comatose and hyperventilating is believed to have severe metabolic acidosis. He is given a solution of M/6 sodium lactate. The favorable effect on the sensorium and respirations which is observed supports the tentative diagnosis.

Similarly, a patient with oliguria is given a "testing infusion" of glucose to determine whether the decreased output is the result of

solution." Some patients suffer from acute dehydration, others from chronic dehydration. In some, the ECF is hypertonic; in others, hypotonic. Potassium depletion or intoxication may also exist. Sometimes there is an associated hyperchloremic acidosis or alkalosis. In each instance the patient will need fluids, but the physician must modify the composition of the hydrating solution so that balance will be re-established safely and surely.

When prolonged parenteral therapy is required, vitamins, calories and amino acids must also be supplied. A deficiency in any essential nutrient will accentuate fluid and electrolyte disturbances and add to the difficulties of management. Positive nitrogen balance cannot be established in the face of a fluid and electrolyte imbalance.

The requirements should be introduced through the gastrointestinal pathway when possible. Proctoclysis, or rectal administration of fluids, electrolytes and nutriment, has little place in current practice. Hypodermoclysis (introduction of solutions under the skin) is likewise little used, except in infants and children or when superficial veins cannot be cannulated.

Generally, the intravenous route is used for parenteral therapy. A No. 20 needle with a short bevel is placed in an arm vein, preferably away from a joint and in such a position that the needle adapter may be taped firmly against the arm. This allows some movement of the arm without dislodgement of the needle. The rate of administration depends on the condition of the patient but should not exceed 500 ml./hr. in the healthy adult.

There are certain hazards to the use of parenteral fluids.

1. Hypertonic or nonelectrolyte solutions given in large amounts by hypodermoclysis result in a rapid distributional shift of ECF into the area of injection. Depletion of ECF may lead to shock.

2. Overloading, or intravenous infusion of fluid too rapidly or in excessive amounts, may result in pulmonary edema or right heart failure, or both.

3. Dehydration from diuresis may result from too rapid administration of hypertonic solutions.

4. Potassium intoxication, with death from cardiac arrest, may result from rapid or excessive infusion of potassium salts. This danger exists especially when urinary output is inadequate.

5 Water intoxication (from giving water without electrolytes, to repair hypotonic dehydration) results from fluid shift from the ECF into the ICF.

Solutions and Routes of Administration

For a list of repair solutions, see Table 8. The simple repair fluids, 5 and 10 per cent glucose, 0.9 per cent saline, 3.0 per cent saline, M/6 sodium lactate and ampules of potassium chloride generally

TABLE 8.—Repair Solutions

Solution	Cations in mEq./L.	Anions in mEq./L.
Glucose in water 5%		
Glucose in water 10%		
Isotonic saline solution (0.9% NaCl)	154 Na	154 Cl
Hypertonic saline solution (3.0% NaCl)	520 Na	520 Cl
M/6 sodium lactate (1.9%)	166 Na	166 mM. lactate
Other:		
Sodium bicarbonate, isotonic (1.5%)	178 Na	178 mM bicarbonate
Darrow's "K" lactate (full strength)	120 Na 35 K	105 Cl
Diluted:		
⅓ Darrow's to ⅔ glucose in water	48 Na 14 K	42 Cl
⅔ Darrow's to ⅓ glucose in water	72 Na 21 K	63 Cl
Ringer's lactate (modified Hartmann's solution)	130 Na 4 K 4 Ca	111 Cl 27 mM. lactate
Amino acid solutions (protein hydrolysates):		
Amigen® (Mead Johnson) (5%)	30 Na	25 Cl
Aminosol® (Abbott)	less than 10 Na	
Travamin® (Baxter)	113 Na	51 Cl
Plasma	142 Na 5 K 5 Ca 2 Mg	103 Cl 27 bicarbonate 5 lactate 2 phosphate

will provide all that is necessary in prescribing specific parenteral therapy. Special solutions (Hartmann's, Darrow's, Ringer's, lactated Ringer's, Butler's polyionic, "gastric replacement," "intestinal replacement," etc.) are more expensive, frequently unavailable and too often are used empirically. The "eponymic" solutions can be used *safely only if the physician knows their composition and makes the selection on the basis of what the patient needs.*

It is illogical to give all dehydrated patients the same "hydrating

Hypertonic fluids need to be given on a twenty-four hour a day basis so that an osmotic diuresis will not result. The purpose of delivery of the infusion into a large vein is to prevent the irritating solution from reaching the vessel wall before it can be diluted with a large quantity of blood. Even under these conditions, the infusion site must be watched for signs of thrombophlebitis, and administration into one vein should not be continued beyond three days. Heparin added to the infusion (25 mg./L.) will help to prevent local thrombosis.

Two thousand milliliters of 10 per cent glucose with 5 per cent amino acids and approximately 5 per cent alcohol will provide close to 2,000 calories.* The solution will have a pH of 5.5 and will supply 60 mEq. of sodium and 30 mEq. of potassium. Two potential complications from such therapy exist. In a few days, a hyperchloremic metabolic acidosis usually develops unless M/6 sodium lactate is also given. If 3,000 ml. of this amino acid, carbohydrate, alcohol solution are given each day, the patient will tend to become dehydrated. To avoid these difficulties, he may be given 500 ml. of 5 per cent glucose and an equal amount of M/6 *sodium lactate along with each 2,000* ml. of the high-calorie fluid mixture. Vitamins, including B complex and C, are added, since they are necessary for the metabolism of carbohydrates and amino acids. If 3,000 ml. of total fluid are to be given in twenty-four hours, the rate is calculated at about 33 drops per minute.

$$\frac{3{,}000 \text{ ml.} \times 16 \text{ drops/ml}}{24 \text{ hr.} \times 60 \text{ min./hr.}} = 33$$

The foregoing cases require only *maintenance*, without *replacement* or *corrective* therapy.

The next degree of complexity will include examples in which it is necessary to supply, not only the *daily metabolic needs*, but also *fluids and electrolytes being lost* from the metabolic pool.

CASE 3.—*Loss of acid gastric juice.*

A 70 kg. man was well until three hours before hospital admission, when he was seized with severe abdominal pain. On admission the abdomen was found to be board-like, and in an upright x-ray film, air was seen beneath the diaphragm. The patient was operated on and a perforated duodenal ulcer closed. Nasogastric suction was used postoperatively.

What fluids will be needed during the first and subsequent postoperative days? As part of the metabolic response to stress, there will be a decreased urinary output of water and sodium with an increased output of potassium for twenty-four to forty-eight hours. Excessive water and salt loads, therefore, cannot be excreted. During this period, he will need about 2,500 ml. of 5 or

* Calculation

200 Gm. glucose (4 calories/Gm.)	=	800 calories
100 Gm. amino acids (4 calories/Gm.)	=	400 calories
100 ml. alcohol (7 calories/ml.)	=	700 calories
Total calories	=	1,900 calories

6. Acidosis may result from giving isotonic saline to replace neutral or alkaline fluids lost from the body. A 0.9 per cent saline solution physiologically has an *acidifying* effect because it contains an excess of Cl ions (155 mEq./L) over Cl ions in the ECF (103 mEq./L.).

7. Pyrogenic reactions (chills and fever) usually are due to improperly prepared solutions or faulty equipment.

8. Bacteremia and shock may result from infusion of contaminated or septic solutions.

9 Thrombophlebitis may result from careless administration, prolonged infusion or hypertonic solutions.

Additional Material

The student frequently asks, "What book can I read?" There are many books available, but none that will supply exactly what the student wants. This subject is not something that can be read and memorized. Each student must learn a common and generally accepted vocabulary and then spend time and intensive effort, individually and in discussion, applying the basic concepts. Once the approach to these problems has been mastered, any book or patient can be used as a pabulum for critical digestion and understanding. Supplemental material in the form of monographs, papers, individual case studies, group discussions, etc, will then make this material an integral part of physician's thinking.

As an aid to the student, some illustrative cases follow:

CASE 1.—*An uncomplicated case.*

A normal, robust 70 kg patient, during the first day after an emergency appendectomy, requires minimal parenteral therapy. He is given 2,000 ml. of 5 per cent glucose to provide for fluid loss through insensible perspiration and urine Electrolytes, vitamins, calories or amino acids need not be given. Oral intake is resumed after the first or second day.

CASE 2.—*A slightly more complex situation.*

A 70 kg. patient has had perforation of the cervical esophagus from endoscopic examination. He will be unable to swallow fluid or food for five or six days, but recovery is otherwise uncomplicated.

The normal daily intake of NaCl (4 5 Gm.) and KCl (3 Gm.) should be provided If 3,000 ml of fluid a day are given as 10 per cent glucose, it will be possible to give 1,200 calories (300 Gm. × 4 calories/Gm.). This is inadequate and the patient will catabolize some body fat and protein. If he is malnourished, a larger caloric intake may be desirable. By the use of a plastic catheter threaded into a large vein, hypertonic solutions can be administered slowly for long periods.

output in the face of the stress reaction, edema and pulmonary congestion will result and may produce fatal complications. (See Chapter 8, p. 181.)

CASE 5.—*Loss of alkaline gastrointestinal fluid*

This patient has had no pre-existing chemical imbalance but has had elective ileostomy and colectomy for chronic ulcerative colitis. He will require enough fluid to provide for insensible loss and urine output each day. After the first day, he will need a daily maintenance dose of potassium (40 mEq.) and NaCl (4.5 Gm.). In addition, he will need replacement, on a volume-for-volume basis, of all gastrointestinal fluid lost. Initially, the main loss will be gastric juice through nasogastric suction; this juice is replaced with isotonic saline solution. After a few days, the patient's ileostomy will begin to function and then most of the fluid loss will be replaced with one part of M/6 sodium lactate and two parts of saline solution (plus 40 mEq. K/L. of electrolyte solution given). If the urinary pH becomes markedly acid or markedly alkaline, the ratio can be corrected. If an excessive amount of chloride is excreted in the urine, electrolyte replacement should be reduced. When urine output increases, ileostomy output decreases and there is evidence of increased absorption of oral fluids, the parenteral therapy may be gradually discontinued.

Now let us turn to more complicated examples in which, in addition to current metabolic needs and current losses, deficits or chemical imbalances exist. These problems may be simple when fluid volume alone is increased or decreased, when tonicity alone is increased or decreased, when a single ion (e.g., potassium) is deficient or when an abnormality in pH alone exists. But the patient can have a combined abnormality of volume, osmolarity and chemical composition and of acid-base balance. Four examples of deficit problems are presented. The first three involve: *isotonic dehydration, metabolic acidosis* and *hypotonicity* (water intoxication), respectively. The fourth case is an example of *combined dehydration, hypotonicity and acidosis*. In order to follow these examples, it may be necessary to make use of the models, symbols and basic concepts and to actually diagram ("gamblegram") each case. This will be left up to the reader.

CASE 6.—*Isotonic dehydration*

A thin man weighing 60 kg. has been vomiting large amounts of bile-stained fluid for two days because of small-bowel obstruction. He appears severely dehydrated. Skin turgor is poor, eyeballs soft and tongue furrowed. He is breathing normally. The pulse is fast, and the blood pressure low. The urine is concentrated, scanty and slightly acid.

It is important, in each instance, to determine whether there is a deficit in water and salt (isotonic dehydration), a deficit largely of water (hypertonic dehydration) or a deficit primarily of salt (hypotonic dehydration). It is also necessary to decide how much of the

10 per cent glucose for metabolic needs, enough 0.9 per cent saline solution to replace the observed loss of acid gastric juice, and added vitamins.

Gastric juice is usually a mixture of parietal (HCl) and mucous cell secretions with more or less saliva, bile, pancreatic juice and succus entericus. It can usually be replaced with isotonic saline on a volume-for-volume basis. A piece of nitrazine paper can be used to test the pH of the gastric aspirate, and the other end of the paper can be used to determine the pH of the urine. If replacement is inappropriate or inadequate, loss of acid juice will lead to a metabolic hypochloremic alkalosis. Under these conditions the urine will be alkaline and the chloride output low (Fantus test).

No KCl is given parenterally during the first twenty-four to forty-eight hours. By the second or third day, if parenteral fluid is still needed, potassium may be given if urinary output is adequate. The normal potassium requirement is about 40 mEq. per day. An additional 30–40 mEq. of potassium is
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body potassium is also replenished. Administration of isotonic saline without potassium will actually lead to an increased potassium loss without improving the hypochloremic alkalosis.

CASE 4.—*Loss of extracellular-like fluid.*

A 70 kg. man has had an extensive burn. Because such an injury provokes a marked stress reaction, there will be water and sodium retention, and overhydration is a real danger. This is especially true in elderly patients and in patients with burns about the face who have sustained damage to the respiratory passages.

No potassium is given in any form during the acute postburn stage. For this reason, fruit juices are forbidden. The ECF potassium must not reach a lethal level (around 10 mEq./L.). Ultimately, potassium may be required; but by the time it is needed, the patient will usually be able to take oral feedings.

In order to replace fluid lost into and from the burn area, and to replace blood sequestered in the burned area (distributional shift) and/or hemolyzed, equal parts of extracellular-like fluid and blood are given. The fluid may be taken orally, if possible, in the form of a solution containing 1 level teaspoonful of salt and 1 level teaspoonful of sodium bicarbonate ($NaHCO_3$) added to a quart of flavored ice water. If the fluid must be given intravenously, then it is made up in a ratio of one part of M/6 sodium lactate to four parts of isotonic saline solution, 1.4 is approximately the ratio of $NaHCO_3$ to NaCl present in the ECF. If saline solution alone is used to replace the ECF, a hyperchloremic metabolic acidosis will result.

How much blood and electrolyte solution should be given? It should be sufficient to maintain: a pulse below 100/minute, blood pressure normal, sensorium clear and urinary output adequate (800 ml./day = 35 ml./hr., or 9–10 drops/min.). If one tries, by forcing fluids, to achieve a high urinary

output in the face of the stress reaction, edema and pulmonary congestion will result and may produce fatal complications. (See Chapter 8, p. 181.)

CASE 5.—*Loss of alkaline gastrointestinal fluid*

This patient has had no pre-existing chemical imbalance but has had elective ileostomy and colectomy for chronic ulcerative colitis. He will require enough fluid to provide for insensible loss and urine output each day. After the first day, he will need a daily maintenance dose of potassium (40 mEq.) and NaCl (4.5 Gm.). In addition, he will need replacement, on a volume-for-volume basis, of all gastrointestinal fluid lost. Initially, the main loss will be gastric juice through nasogastric suction; this juice is replaced with isotonic saline solution. After a few days, the patient's ileostomy will begin to function and then most of the fluid loss will be replaced with one part of M/6 sodium lactate and two parts of saline solution (plus 40 mEq. K/L. of electrolyte solution given). If the urinary pH becomes markedly acid or markedly alkaline, the ratio can be corrected. If an excessive amount of chloride is excreted in the urine, electrolyte replacement should be reduced. When urine output increases, ileostomy output decreases and there is evidence of increased absorption of oral fluids, the parenteral therapy may be gradually discontinued.

Now let us turn to more complicated examples in which, in addition to current metabolic needs and current losses, deficits or chemical imbalances exist. These problems may be simple when fluid volume alone is increased or decreased, when tonicity alone is increased or decreased, when a single ion (e.g., potassium) is deficient or when an abnormality in pH alone exists. But the patient can have a combined abnormality of volume, osmolarity and chemical composition and of acid-base balance. Four examples of deficit problems are presented. The first three involve: *isotonic dehydration, metabolic acidosis* and *hypotonicity* (water intoxication), respectively. The fourth case is an example of *combined dehydration, hypotonicity and acidosis.* In order to follow these examples, it may be necessary to make use of the models, symbols and basic concepts and to actually diagram ("gamblegram") each case. This will be left up to the reader.

CASE 6.—*Isotonic dehydration.*

A thin man weighing 60 kg. has been vomiting large amounts of bile-stained fluid for two days because of small-bowel obstruction. He appears severely dehydrated. Skin turgor is poor, eyeballs soft and tongue furrowed. He is breathing normally. The pulse is fast, and the blood pressure low. The urine is concentrated, scanty and slightly acid.

It is important, in each instance, to determine whether there is a deficit in water and salt (isotonic dehydration), a deficit largely of water (hypertonic dehydration) or a deficit primarily of salt (hypotonic dehydration). It is also necessary to decide how much of the

dehydration is in the ECF space and how much involves the whole body water. The more acute the illness, the more likely that the deficiency is only in the ECF, especially when vomiting and/or diarrhea have contributed to the abnormality.

Tonicity and pH of the ECF are thought to be near normal in this patient, and this impression is confirmed by the serum chemistry reports (given in mEq./L.). Na 145, Cl 100, CO_2- combining power 27 and K 4.

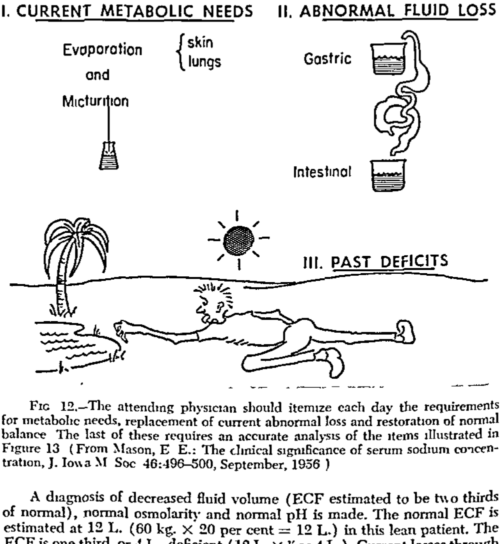

FIG. 12.—The attending physician should itemize each day the requirements for metabolic needs, replacement of current abnormal loss and restoration of normal balance. The last of these requires an accurate analysis of the items illustrated in Figure 13. (From Mason, E. E.: The clinical significance of serum sodium concentration, J. Iowa M. Soc. 46:496–500, September, 1956.)

A diagnosis of decreased fluid volume (ECF estimated to be two thirds of normal), normal osmolarity and normal pH is made. The normal ECF is estimated at 12 L. (60 kg. × 20 per cent = 12 L.) in this lean patient. The ECF is one third, or 4 L., deficient (12 L. × ⅓ = 4 L.). Current losses through nasogastric suction are estimated at 3,000 ml./24 hr. of mixed juices having a pH of 7.4 when tested with nitrazine paper and a composition approximately the same as ECF. The 24-hour metabolic needs are 2,000 ml. for urine and insensible losses. The total fluid requirements, therefore, are 4,000 + 3,000 +

2,000 = 9,000 ml., of which 7,000 ml. is similar to ECF and can be replaced with an ECF-like solution of four parts isotonic saline solution to one part M/6 sodium lactate. The remaining 2,000 ml. may be supplied as glucose (5 per cent) in water.

This is only an estimate. The response to treatment, including operation, will require alteration of the prescription. We can estimate that initially the infusion will have to run at about 110 drops/minute

$$\frac{9{,}000\text{ ml.} \times 10\text{ drops/ml.}}{24\text{ hr.} \times 60\text{ min./hr.}} = 110\text{ drops/min.}$$

Potassium, 40 mEq. (3 Gm. KCl) will be added to each liter of electrolyte solution, but only after urinary output has been established.

The fluid order for the first twelve hours is written:

1,000 ml. isotonic saline solution
500 ml. M/6 sodium lactate
1,000 ml. isotonic saline solution
1,000 ml. 5 per cent glucose in water
1,000 ml. isotonic saline solution (with 3 Gm. KCl added)
250 ml. M/6 sodium lactate

The rate of infusion is adjusted to about 110 drops/minute. A record is kept of the approximate amount given each hour and the volume of urine excreted each hour.

The physician must re-evaluate the patient's condition frequently and make such alterations as seem indicated on the basis of the patient's response to therapy: symptoms, appearance, urinary output, urine specific gravity and the pH, and the volume of gastric and intestinal juice aspirated.

In the above prescription, half the requirement of water for daily metabolic needs will have been met, the remainder can be supplied in the second 12-hour period. About 3,000 ml. more of ECF-like solution is needed to balance losses. This may be given in the next twelve or twenty-four hours, depending on the patient's response. Additional KCl will be added when urinary output is normal.

CASE 7.—*Metabolic acidosis.*

A man weighing 60 kg. has had diarrhea for two days and his physician has administered isotonic saline solution (with KCl) intravenously in amounts equal to the liquid stools which have been passed. In addition, the patient has received 5 per cent glucose and the urine output has remained adequate. He is not thirsty, and skin turgor is normal. It is observed that he is somewhat lethargic and is breathing deeply and at an increased rate.

It is concluded from the history, physical findings and some simple bedside laboratory tests (urinary pH, urine chloride, urine specific gravity) that the patient is not dehydrated and that he has normal osmolarity, but that he is in severe acidosis as a result of (1) a loss of alkaline gastrointestinal fluid and (2) its replacement by isotonic saline solution, which is physiologically an acidifying solution.

The blood chemistry values (in mEq./L.) are: Na 135, K 5, Cl 120 and CO_2 12. The patient needs sufficient M/6 sodium lactate to change his base

carbonate from 12 to 27, or an increase of 15 mEq./L.; and he needs to have this change made in his ECF, which is estimated to be about 12 L. He therefore requires 12 × 15 mEq. of Na^+, or 180 mEq. Sixth molar sodium lactate contains 166 mEq. Na/L.; so 1,100 ml. is administered. Current losses are replaced with a solution containing isotonic saline solution and M/6 sodium lac-

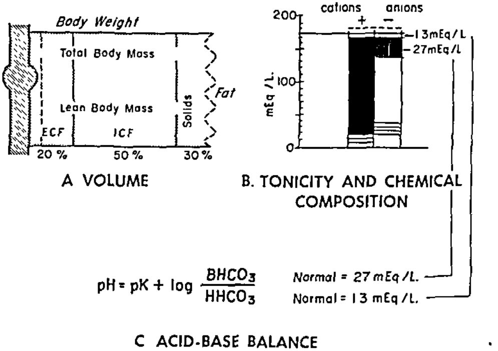

FIG. 13.—To restore a patient to normal balance, it is necessary that the physician know qualitatively and quantitatively the abnormalities that are present. A tripartite diagnosis should be made concerning (1) volume, (2) tonicity and chemical composition and (3) acid-base balance. A, body weight is shown to include a vari- [illegible] $BHCO_3$ of 27 mEq./L. is normal. C, the Henderson-Hasselbalch equation is useful, with diagram B, for interpreting acid-base changes. (Adapted from Mason, E. E.: The clinical significance of serum sodium concentration, J. Iowa M. Soc. 46:496-500, September, 1956.)

tate in a ratio of 2 to 1, with 3.0 Gm. KCl/L. and the same amount of fluid for daily metabolic needs that he has been getting. A *diagnosis ex juvantibus* is then made, and the treatment is modified according to the progress.

CASE 8.—*Hypotonicity (water intoxication).*

The patient is an obese woman weighing 250 lb. It is estimated, from her height and build, that her normal weight should be about 132 lb., or 60 kg., which represents her "lean body mass." This calculation is necessary because fat contains little water and is relatively inactive metabolically.

A long operation for carcinoma of the breast is performed on the patient. During the night she has convulsions, and it is feared that she may have had a cerebrovascular accident.

She has lost a great deal of extracellular fluid from the wound both during and after the operation. Glucose solution, 4,000 ml., without electrolytes has been given postoperatively. Blood chemistry values (in mEq./L.) are: Na 125, K 2.5, Cl 85 and CO_2 17. A diagnosis is made of normal or slightly increased fluid volume, severe hypotonicity (water intoxication) and normal acid-base balance.

The patient is given 200 ml. of 3 per cent saline solution over a period of four hours and responds satisfactorily. Larger amounts of hypertonic saline solution are not usually given in any one four-hour period. If more is needed, the blood chemistry should be rechecked before the infusion is repeated. It is decided that no more fluids will be needed the next day. The patient will excrete the excess water and should soon be able to take fluids by mouth.

CASE 9.—*Combined dehydration, hypotonicity and acidosis.*

Let us suppose that the above obese patient develops severe diarrhea on the third postoperative day. She receives a repair solution consisting of equal parts of glucose and saline in inadequate amounts because no one knows exactly the volume of the liquid stools. The patient is found in coma. Her eyeballs are soft, skin turgor poor, pulse fast, blood pressure low and respirations rapid. The lungs are clear on auscultation.

The urinary output has been 400 ml. in the preceding 10 hours. Specific gravity is 1.012, and reaction is acid. The Fantus test shows 1 Gm. Cl/L. The report from blood chemical analysis shows (in mEq./L.): Na 125, K 2.5, CO_2 12 and Cl 96. The tentative fluid diagnosis is: dehydration, hypotonicity and acidosis.

As previously stated, the patient's normal lean body mass is estimated to be 60 kg., giving a normal ECF volume of 12 L. The volume of her ECF is now estimated to be one third reduced, so 4 L. of ECF-like fluid is needed. The estimated ECF [illegible] and Cl levels suggest [illegible] tonicity. The Na^+ defi[illegible] = 240). The continuing loss of fluid through diarrhea is now being measured by use of a rectal tube connected to a bottle at the bedside. It appears that about 4,000 ml. will be lost in the next twenty-four hours. The metabolic needs for the day will be about 2,000 ml.

To summarize:

1. Past deficit in volume of ECF = 4,000 ml.
2. Past deficit osmolarity in remaining ECF = 240 mEq. Na.
3. Past deficit of Cl. = 84 mEq. (103 − 96 = 7 mEq./L., or total deficit of 7 × 12 L. = 84 mEq.). This is not as great as the Na^+ deficit (20 mEq./L.) because the patient has received an excess of Cl^- in the saline infusions.
4. Current loss = 4,000 ml.
5. Metabolic water = 2,000 ml.
6. Total amount of fluid = 10,000 ml.

The volume of fluid to be replaced is 10,000 ml. The hypotonicity can be corrected by simply placing the 240 mEq. Na^+ and 80 mEq. of Cl^- in the 2,000 cc. of metabolic fluid. It is therefore unnecessary to give hypertonic fluid. This means that about 6,000 ml. of the repair fluid should be similar to alkaline digestive secretions (isotonic saline : M/6 sodium lactate :: 2 : 1),

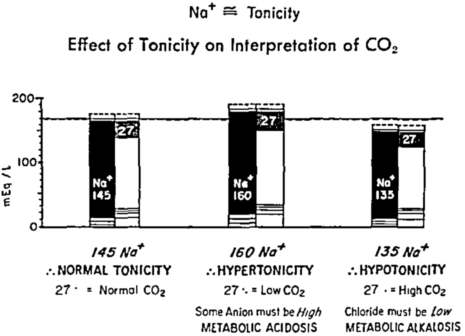

FIG 14.—These bar graphs show the concentration of cations and anions in the ECF The total number of particles (cations, anions and nonionized substances) in a solution governs its tonicity. Since sodium (in black) is the predominant cation in extracellular fluid and since there are usually few nonionized solutes, variations in sodium concentration indicate variations in tonicity. Since any loss or addition of water will affect the concentration of all ions, it is essential that tonicity be estimated before the chemical composition is analyzed. (Adapted from Mason, E. E.: The clinical significance of serum sodium concentration, J Iowa M. Soc. 46·496-500, September, 1956)

and 4,000 ml. should be similar to ECF (isotonic saline : M/6 sodium lactate :: 4 · 1) Again, this is a pretreatment estimate for starting therapy and for determining the type and rate of fluid administration. As the day and therapy progress, it may be necessary to modify this estimate.

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CHAPTER 5

Nutritional Balance

THE PROBLEMS of nutrition are not peculiar to surgery but encompass all of medicine. In surgical practice, however, many conditions are associated with actual or threatened nutritional depletion. Often the normal routes for alimentation are unavailable and artificial feedings are required.

The importance of food in all phases of management should be considered. Throughout the course of the patient's illness, and particularly following the period of stress, nutriment is required for repair of tissue and support of metabolism. In depleted states the homeostatic mechanisms are seriously compromised, disposing the patient to many complications, with delayed convalescence or even death. It is in keeping with the admonition to "make the patient safe for surgery" that every effort be expended to establish and maintain that state of body economy which is as near normal as possible in order to minimize the hazards which trauma, disease and surgical treatment itself impose.

The problems of nutritional sustenance are not difficult in the young and vigorous patient who is subjected to an operation of moderate severity, provided that prolonged suspension of gastrointestinal function does not occur. On the other hand, patients who have suffered serious illness and nutritional depletion require special care to insure sufficient intake of the elements necessary for tissue repair and recovery.

Factors Necessary for Normal Nutrition

Protein, carbohydrate and fat, together with water, salts and essential vitamins, must be supplied in an appropriate balance or admixture; and the diet must contain sufficient calories to maintain

health There is a close interrelationship between the many items in the diet: deficiency in one category may lead to increased metabolism or loss of other substances. All of these constituents are available for oral, tube and intravenous administration. At the present time, intravenous solutions of fat are not widely used because they provoke a relatively high incidence of pyrogenic reactions. It is likely that this problem soon will be solved.

Proteins are necessary in almost every aspect of bodily function. Ingested proteins are acted upon by the digestive processes and broken down into their primary constituents, amino acids, which are absorbed in the small intestine and carried by the portal circulation to the liver. Some amino acids pass through the liver unchanged and are utilized for repair, regeneration or growth of tissues or formation of enzymes and hormones. Some are broken down in the liver (deaminized), where the amino groups are split off and converted to urea, which is excreted in the urine. The remainder is oxidized for energy or converted into glucose.

Except for a negligible loss through the saliva, sweat and hair, nitrogen normally leaves the body only in the urine and feces. The protein balance of the body represents the difference between nitrogen intake and nitrogen output, which can be determined by balance studies. When intake of nitrogen equals output, the individual is said to be in zero balance; when loss exceeds intake, he is in negative balance; and when intake exceeds loss, he is in positive balance.

As noted above, amino acids can be converted to several metabolic groups according to the specific need at the moment. However, in order for the body to utilize these substances in protein synthesis, all the essential amino acids must be present in ample supply. These include methionine, valine, leucine, isoleucine, threonine, phenylalanine, tryptophan and lysine. When one or more of the essential amino acids is lacking, the body is unable to utilize any of the other amino acids in synthesis of protein, and they are lost as nitrogen in the urine. Not only must these elements be present in sufficient concentration, but they must be present at the same time if they are to be used.

The dangers of protein depletion include:

1. Anemia and constriction of the total blood volume (hypovolemia), increased susceptibility to shock, poor response to anesthesia and operative trauma.
2. Defective fibroplasia and delayed wound healing, increased incidence of wound disruption, decreased formation of callus

after fracture and a predisposition to develop decubitus ulcers.

3. Hypoproteinemia; subcutaneous, submucosal and pulmonary edema; delayed gastrointestinal motility; anastomotic obstruction and leakage.
4. Deficiency in antibodies, lowered resistance to infections.

In health and when there is an adequate caloric intake, protein in the amount of 1 Gm./day/kg. body weight is sufficient to maintain a state of nitrogen balance in the adult. Unless caloric requirements are met, however, protein will be utilized for energy. Carbohydrates and fats are the main sources of energy in the normal diet and provide 4 and 9 calories/Gm., respectively.

Under conditions in which intake, assimilation, utilization or retention of food is disturbed, the protein requirement may amount to 3–5 Gm./kg./day before positive nitrogen balance is restored. If possible, the dietary requirements should be given in the form of natural foods through natural routes. When this is impossible, artificial routes and formulas may have to be utilized. These will be described later.

Carbohydrates, after digestion to monosaccharides (e.g., glucose, fructose, galactose), are absorbed and carried to the liver, where they are all converted to glucose and oxidized, polymerized to glycogen, or diverted for protein or fat production. Protein, and possibly fat, is also converted to glucose and glycogen in the liver. Part of the glycogen is stored in the liver, and the remainder is stored in the body tissues, chiefly as muscle glycogen.

Hepatic glycogen stores are relatively small and require frequent replenishment. The normal adult liver has been estimated to have a maximum carbohydrate storage capacity of only 150 Gm. (600 calories), which is readily available but quickly depleted under stress.

Carbohydrates normally provide a substantial portion of the daily energy requirements. When the caloric intake becomes inadequate, the body is forced to convert exogenous, or, if necessary, endogenous, protein into carbohydrate to meet caloric needs. The physician must be acutely aware of these facts and exploit the protein-sparing action of carbohydrate to its maximum. An adequate carbohydrate intake is also necessary for the complete metabolism of fat and the prevention of ketosis.

Fat also has a protein-sparing action and normally is a rich source of calories Storage in fat depots depends, in large part, on the balance between caloric intake and energy output. Body fats are

formed from ingested fat and carbohydrate, as well as from protein, when caloric intake is in excess of physiologic needs.

The role of the liver in fat metabolism must also be considered. When the intake of carbohydrate and protein is deficient, an excessive deposition of fat occurs in the liver, lowering its metabolic efficiency and rendering it susceptible to injury from anoxia or toxic agents or depressant drugs. This is the basis for the practice of "fortifying the liver" preoperatively with a high-carbohydrate, high-protein and low-fat intake.

In health, a balanced diet supplies vitamins in adequate amounts. In deficiency states, these substances are often required as supplements along with an increased food intake In surgical patients, deficiencies in vitamin B complex and vitamin C may occur, since these factors cannot be stored in reserve depots. When fat absorption is impaired (as in common bile duct obstruction), deficiencies in the fat-soluble vitamins A, D and K are to be anticipated. The role of vitamin K in the formation of prothrombin will be discussed later.

The vitamin B complex exerts widespread effects on many tissues and organs. There is a close interrelationship between certain fractions and the metabolism of carbohydrates and protein. Thiamine is required *in excess when carbohydrate intake is increased. Niacin, riboflavin* and other factors should also be given in moderate excess in malnutrition. The role of other factors (B_{12}, folic acid) in hemopoiesis should be kept in mind.

Vitamin C is needed for tissue repair and wound healing. In deficiency states, the basic defect in wound healing appears to be related to failure of formation of normal intercellular ground substance of connective tissue, and maturation of collagen. Under these conditions, wound healing is delayed, tensile strength is below normal and scar-tissue formation is defective.

Vitamin C may also be involved in the synthesis of adrenal cortical hormones, but the exact relationship has not been clarified.

In the well-nourished patient, the stores of vitamins A and D are generally adequate. The influence of vitamin A on epithelization must be recognized, but supplementation is rarely required. An increased intake of vitamin D is unnecessary except during the growth period. Adverse effects from excessive vitamin D therapy, in the form of renal calculi in bedfast or immobilized patients as a result of excessive calcium excretion, have been reported.

Depletion of plasma prothrombin may result from failure of ab-

sorption of vitamin K due to absence of bile salts in the intestine (obstructive jaundice) or from destruction of the vitamin K-producing bacteria of the gut by antibacterial drugs. In these situations, correction of the prothrombin deficiency can be achieved by the administration of synthetic vitamin K preparations.

Causes of Malnutrition

The factors leading to impaired nutrition are varied and complex. They may be analyzed by asking the following questions:

1. *Is there simple anorexia with failure to eat?*

Simple anorexia with failure to eat is a common difficulty which may lead to serious accumulated deficits. It may be unsuspected because there is always a tendency to look for more complex disturbances. The aged person, who lives alone and prepares his own food, often is so afflicted. A vicious cycle may be set up, leading to progressive nutritional deterioration. Although economic factors may be involved, one must also bear in mind the changes which are incident to the aging process, such as diminution of the sense of taste, decrease in the secretion of saliva, lack of teeth or the presence of ill-fitting dentures, and the tendency to psychic depression or disinterest in all activity, including eating.

There appears to be no justification for the widespread belief that the appetite always exerts a beneficial regulating effect on metabolic needs. Often if one awaits spontaneous return of appetite, there will be continued malnutrition and delayed convalescence. For this reason, it may be necessary to "prime the pump" by getting the patient started on the road to nutritional reconstruction before his appetite returns and finally takes over as the regulator of his nutritional needs.

2. *Is there an accelerated loss of protein?*

Patients with extensive burns, chronic infections, ulcerative colitis, and malignant diseases usually suffer abnormal losses of protein. Weight loss, and specifically loss of lean tissue mass, is the most important indication of nutritional depletion. While it may be impossible to reverse such a trend preoperatively, the surgical attack can usually be delayed for a short time, during which a hyperalimentation regimen is instituted. By this means, it is often possible to restore a state of relative positive nitrogen balance and thereby decrease the risk from operation.

3. *Is there an acceleration of metabolism?*

Hypermetabolism (e.g., hyperthyroidism) increases the patient's caloric needs; and if intake is inadequate, both endogenous and exogenous protein will be utilized as fuel, thus producing a state of negative nitrogen balance. The problem here is to augment caloric, protein and vitamin intake as well as to control the hypermetabolic state.

4. *Is there a suspension of gastrointestinal tract activity?*

This condition occurs temporarily following injury or operation. When there is prolonged interference with alimentary tract function, as in obstruction of the stomach or bowel (Fig. 15), special measures, such as tube and parenteral feedings, are necessary to correct deficiencies.

5. *Is there poor utilization of foodstuffs?*

Poor utilization of foodstuffs is a less common but an important cause of nutritional depletion. It may result from loss of part of the absorptive surface of the small intestine from disease (e.g., regional enteritis) or resection, from short-circuiting of food through external or internal fistulas, or from deficient secretion of the digestive fluids of the pancreas or liver. Except under these conditions, nutritional failure generally can be ascribed to other causes

Changes with Malnutrition

Regardless of the cause, nutritionally depleted patients are likely to exhibit one or more of the following alterations:

1. Weight loss, decrease in fat-tissue stores and wasting of lean tissue mass (muscle) generally imply an inadequate food intake, with deficiency in calories and protein, and a state of negative nitrogen balance for a considerable period of time. There may be specific vitamin deficits as well, each with more or less clearly defined clinical signs.

2. Chronic malnutrition is nearly always associated with dehydration, anemia, hypoproteinemia and hypovolemia. Loss of body water may be regarded as a compensatory mechanism for the maintenance of normal plasma osmotic tension when the plasma protein content is reduced. There is a concomitant reduction in the total blood volume (hypovolemia) leading to a condition sometimes called "chronic shock." Hemoglobin deficiency and hypoproteinemia regularly

Fig 15.—Severe malnutrition due to prolonged chronic partial intestinal obstruction resulting from intraperitoneal adhesive bands. The patient had had numerous operations for pelvic inflammatory disease in early adult life, followed by four operations for intestinal obstruction Note the extreme degree of muscle wasting and loss of subcutaneous fat, as well as the abdominal distention and loops of bowel visible through the thin abdominal wall. Radiographic studies confirmed the diagnosis of chronic partial small-bowel obstruction. After intensive preoperative preparation, a segment of scarred obstructed ileum was removed and continuity established by anastomosis The postoperative course was complicated by thoracic empyema, but ultimately the patient made a good recovery and her weight increased from 78 to 144 pounds in three months. At a five-year follow-up, she was found to be well.

accompany hypovolemia under conditions of malnutrition. The laboratory findings, including hemoglobin and red blood cell counts, plasma proteins and hematrocrit values in hypovolemia may be relatively normal. Later, when water, salt and volume deficits have been restored, these values are found to be lower than normal. Blood volume determinations, using Evans blue dye (T-1831), or radioisotope technics, are helpful in estimating replacement needs, but they are not essential to successful management.

Treatment of Malnutrition

Priorities in the treatment of nutritionally depleted patients are:

1. Restoration of fluid and electrolyte balance
2. Correction of anemia and hypoproteinemia
3. Restoration of normal blood volume
4. Reversal of negative nitrogen balance
5. Provision of an adequate vitamin intake

In the restoration program the need for water and electrolytes is apparent Detailed consideration has been given to this subject in Chapter 4, on Fluid and Electrolyte Balance, but a few points should be re-emphasized. The need for fluid and electrolytes is relatively greater in the depleted infant or child than in the adult. The need for water is increased when the concentrating power of the kidney has been lost When the protein intake is augmented, extra water for renal function is needed, owing to increased urea formation. Likewise, when salt intake is increased, the need for water is increased.

Transfusions of whole blood are essential for the rapid correction of deficits in hemoglobin, plasma protein and blood volume. While it may appear that plasma transfusions should be adequate, the fact that a deficiency in hemoglobin is a constant feature of the systemic depletion of body proteins emphasizes the need for whole blood. Both blood and plasma will spare exogenous and endogenous protein, but neither will directly provide significant amounts of amino acids for body metabolism. The use of either plasma or blood for the repletion of protein stores other than those of the vascular compartment is expensive and inefficient and is better attained by feeding the patient.

After fluid and electrolyte deficits have been corrected, daily transfusions of 500–1,000 ml. of whole blood may be given, depending on the response to treatment and the severity of the reduction in

hemoglobin, plasma protein and blood volume. While it is usually desirable to restore normal levels rapidly, this may be dangerous in patients who are severely depleted or who have associated cardiac or pulmonary disease. It is estimated that in the adult, 500 ml. of blood will raise the hemoglobin level about 0.5 Gm. Generally, 1,000–2,000 ml. of whole blood will suffice, except when there has been gross loss of blood. The response to administration of blood, in terms of the hemoglobin, red cell count and hematocrit, should be carefully followed. Complete restoration to normal values may be unnecessary or inadvisable, and operation can usually be undertaken when the hemoglobin level has reached about 12 Gm. and the red cell count is 4,000,000.

As noted above, the correction of protein depletion of the plasma and tissue is not simply a matter of giving blood transfusions. It has been shown that a reduction in the plasma protein signifies a thirty-fold reduction in tissue protein stores. These can be reconstituted only by a proper feeding regimen.

When there is need for augmentation of the protein intake, consideration must be given to meeting this need by use of special diets, tube feedings or intravenous feedings singly or in combination, according to the circumstances. If the patient cannot take the prescribed diet, high-protein formula feedings may be given orally or through an inlying nasogastric tube (polyethylene type). Even when there is partial obstruction of the outlet of the stomach, a sufficient amount of liquid formula may pass into the small bowel to bring about reversal of nitrogen balance and weight gain. When an obstruction precludes any type of feeding from above, it may be necessary to establish a temporary (or permanent) route for artificial feeding. This may be a gastrostomy (feeding tube in the stomach) or a jejunostomy, according to the location of the obstruction.

The actual intake in terms of carbohydrates, proteins and fat, together with the total caloric value and the patient's weight, should be charted daily on a metabolic record. It must be realized that there is often a great discrepancy between the amount of food which the patient is served and the amount of food which he ingests. The patient's expected weight, the degree of hypermetabolism and the levels of the circulating proteins should be gauged, and from these values the caloric and protein goals necessary for repletion may be estimated. If it is apparent that through his own efforts the patient will be unable to meet these goals, supplementation is indicated.

The caloric and protein goal must be individualized. In depleted patients it averages about 3,000 calories and 150–200 Gm. of protein per day. If necessary, 5,000 or more calories, and up to 500 Gm. of protein daily may be administered, but a sustained intake of this order is difficult to maintain. The usual division of calories is carbohydrate, 65 per cent; protein, 25 per cent; fat, 10 per cent. In general, the sicker the patient, the lower will be his tolerance for fat and the greater will be his need for protein.

Failure to correct the nutritional problem may be due to the following:

1. Dependence on the appetite of the sick patient
2. Diet not specified
3. Diet not ingested
4. Abnormal losses
5. Defect in assimilation

The type of food offered to the patient should be determined according to his nutritional state, the function of his gastrointestinal tract and his interest in and ability to take food. Insofar as possible, he should be given food which is similar to his daily pattern. It is neither necessary nor desirable to provide luxuries for the patient who has never had them; but it is important that the food provided be of good quality, palatable, hot when intended to be, and attractively served.

The types of diets offered are based on normal requirements. Diets may be varied according to texture or consistency, energy content and individual constituents. The standard hospital diets are:

Light liquids	Soft or bland diet
Full liquids	General diet

The *light liquid* diet consists of tea, coffee, broth and fruit juices, with or without added sugar. This diet is nutritionally incomplete and is intended only to re-establish the oral intake of fluid, electrolytes and carbohydrates.

The *full liquid* diet includes milk, modified and enriched with dried milk proteins, egg, malt and cereals. Ice cream and plain-flavored gelatin are items which enhance this diet. Puréed vegetables and fruit pulp added to milk or meat stock are also used. Frequent small feedings may be given to meet the patient's requirements.

The *soft* or *bland* diet is ordered as strength and ability to take food returns. The amount and frequency of the feedings depends on

the patient's condition. Milk and its many modifications, eggs in almost any form, finely divided meats, puréed fruits and vegetables, and soups or soufflés are admissible.

Progression to a *general* diet or a normal meal pattern is the next step. Fruits and vegetables, cooked and eventually raw, as well as bread and cereals as they occur in the normal dietary, should be used.

TABLE 9.—TUBE FEEDING FORMULA *

CONSTITUENTS	AMOUNT	PROTEIN (GM.)	FAT (GM.)	CARBOHYDRATE (GM.)	CALORIES	SODIUM (MG.)
Sweet whole milk	1,800 ml.	63	70.2	88.2	1,234	900.0
Dextri-Maltose ®	90 Gm.	....	....	90.0	351	756.0
Dextrose	150 Gm.	....	....	150.0	600	1.5
Alacta ®	162 Gm.	53.5	19.4	75.3	697	842.4
Total	1,920 cc. or 2 qts.	116.5	89.6	403.5	2,882	2,500.0
Composition/100 ml.	... .	6	4	21	150	....
Caloric value	About 1.5/ml.	....	....	....	....	....

Vitamins added routinely:

10 gtt. Fer-In-Sol ®	Approx. 9.4 mg. elemental iron
3 gtt. Aquasol	Approx. 4,000 I.U. vitamin A; 800 I.U. vitamin D
2 t. vitamin B syrup	Approx. 40 mg. niacin, 4 mg. thiamin; 3 mg. riboflavin
100 mg. ascorbic acid	2 T. vanilla

* Courtesy of Dr. Kate Daum, Department of Nutrition, College of Medicine, State University of Iowa.

Vitamin supplements should be given as medication when a deficiency exists or is suspected. Synthetic, high-potency preparations are useful at this time. As soon as possible, vitamin preparations containing some crude fraction of liver, rice or yeast should be added to the diet.

TUBE FEEDINGS.—Formula feedings (Table 9), introduced through a nasogastric tube or artificial stoma, may be necessary to augment intake or provide complete nutrition. The base, or carrier, of these formulas usually is whole or skim milk. Additional protein can be supplied by adding dried-milk powder, egg or protein hydrolysates. Calories are furnished by glucose, maltose, sucrose, cream or emulsified vegetable fats. Water-miscible vitamins A and D, vitamin B complex and ascorbic acid are also added.

The caloric and protein goal must be individualized. In depleted patients it averages about 3,000 calories and 150–200 Gm. of protein per day. If necessary, 5,000 or more calories, and up to 500 Gm. of protein daily may be administered, but a sustained intake of this order is difficult to maintain. The usual division of calories is carbohydrate, 65 per cent, protein, 25 per cent; fat, 10 per cent. In general, the sicker the patient, the lower will be his tolerance for fat and the greater will be his need for protein.

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The *soft* or *bland* diet is ordered as strength and ability to take food returns. The amount and frequency of the feedings depends on

balance. Each liter will provide about 50 Gm. of amino acids. At least 2–3 L. daily are required, and some patients will not tolerate this quantity of fluid.

2. Their administration often results in some degree of satiety with decreased ability to take oral feedings. In other words, the hydrolysates given intravenously may "spoil the appetite."
3. Unpleasant side reactions may occur during administration. These include a sensation of heat, fever and abdominal cramps. These reactions are infrequent if the solutions are given slowly (e.g., 1,000 ml. over a period of three hours).
4. Thrombosis of the veins used for infusion sometimes occurs, owing to added glucose and alcohol (hypertonic solution) rather than to the hydrolysate per se. If the solution infiltrates subcutaneously, there may be a severe local reaction, with tissue necrosis and sloughing.
5. Unless adequate calories are provided, the amino acids are metabolized to meet energy requirements rather than to reconstitute proteins.
6. The protein hydrolysates are excellent media for bacterial growth. While the intravenous preparations are available in sterile sealed containers, they are easily contaminated. They must be used immediately after being opened, and they must not be given if cloudy or otherwise altered.

Despite these limitations, the intravenous protein hydrolysates are valuable for short-term nutritional sustenance if little or no food can be taken through normal channels or if continued protein catabolism threatens to prolong or endanger the patient's recovery. They are intended primarily to prevent or reverse protein losses rather than to establish protein anabolism.

It is probable that the administration of protein in any form during the adrenocorticoid phase of the stress reaction (first three or four days after operation) is unnecessary and wasteful. During this period the patient's fluid, electrolyte and energy needs can be met; but little can be accomplished by giving amino acids intravenously, because parenteral nitrogen is excreted almost quantitatively in the urine.

The problem is quite different, however, when the patient is unable to resume oral feedings after the first phase of convalescence has passed. Now he needs protein for tissue repairs; and unless it is available from exogenous sources, it will be derived from endogenous sources. In order to prevent or to minimize the depletion of endog-

Tube feedings should be used with care in patients who give a history of poor food intake. Abdominal discomfort, cramps and diarrhea may follow formula feedings, especially if the amounts administered are large or the concentration of carbohydrate or fat is high. Feedings are given in small amounts initially (50–150 ml.) at one or two hour intervals, or by slow drip if desired. The volume can be increased over a period of days until the desired intake is attained. Additional water and electrolytes may be added to the formula or introduced separately through the tube. The average bed patient tolerates about 250–300 ml. at a single feeding.

Patients receiving tube feedings should be kept under careful surveillance because the incidence of complications is relatively high. Sometimes the mixture, which is a good culture medium, is contaminated with pathogenic bacteria and causes severe gastroenteritis. All feeding formulas must be carefully prepared and stored under refrigeration. Diarrhea may result from mixtures which contain too high a content of fat or easily fermentable sugars. Overloading of the alimentary canal is also a common cause of abdominal cramps and frequent bowel movements.

Administration of tube feedings to patients who are debilitated or who exhibit a lowered level of consciousness (such as those with head injuries) increases the danger of aspiration pneumonitis from vomiting. Often these are the very patients who require augmentation of their nutritional intake, and usually they respond favorably to tube feedings. However, when the feedings are pushed too vigorously or when motility of the gastrointestinal tract is inadequate to propel the feedings, retention and vomiting occurs. Under these circumstances, the mixture may be sucked into the tracheobronchial tree, where it causes severe or fatal obstructive and inflammatory changes unless removed immediately by tracheobronchial catheter suction or bronchoscopy.

Intravenous Feedings.—Solutions of amino acids for intravenous use are prepared by acid or enzymatic hydrolysis of proteins such as casein, fibrin, bovine plasma, lactalbumin and liver. All these solutions are of excellent biologic value and can supply necessary amino acids for protein anabolism.

There are several limitations to the use of intravenous protein hydrolysates:

1. The low concentration of amino acids (5–10 per cent) requires administration of large fluid volumes to attain positive nitrogen

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enous stores when it appears that administration through the gastrointestinal tract is to be delayed, protein may be provided in the form of intravenous protein hydrolysates. Although these preparations will not maintain or restore the patient to a state of positive nitrogen balance, they will usually lessen the degree of negative balance and thus may hasten convalescence. In this regard, Elman has pointed out that "half a loaf is better than none."

When intravenous protein hydrolysates are given, the caloric requirements are met by adding glucose and, if desired, alcohol to the solution Most commercial hydrolysates contain 5 per cent glucose, and the solution can be fortified with glucose to a concentration of 10–15 per cent. Ethyl alcohol in 5–7 per cent concentration may also be added One milliliter of absolute alcohol provides about 7 calories. The nonprotein caloric value of a liter of 5 per cent protein hydrolysate, containing 5 per cent glucose and 5 per cent alcohol, is about 500 calories, or 200 calories from glucose and 300 calories from alcohol. At least 3 L. of such a solution is required to meet basal needs, or 1,500 calories and 150 Gm. of protein Increasing the glucose content of the solution increases the utilization of the amino acids as protein but also increases the tendency to local venous thrombosis.

The necessary daily vitamin supplements can also be added to the intravenous solution. The requirements are met with the administration of: ascorbic acid, 25 mg.; thiamine hydrochloride, 50 mg.; riboflavin, 25 mg., nicotinamide, 100 mg., and vitamin K, 2 mg.

The sodium chloride content of most of these preparations is under 2 Gm./L. The need for potassium during the period of parenteral protein feeding and tissue repair must also be considered and, if necessary, added to the infusion.

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immediate responses to injury will vary according to different mechanisms concerned in their production. Blalock (1934) proposed a classification of shock based on the predominant physiologic mechanisms involved:

1. *Neurogenic shock*—vasodilatation due to diminished vasoconstrictor tone (spinal anesthesia, vasovagal collapse, common faint). These reactions are usually transitory and generally incapable of sustaining a shock state. The conditions in this group have been called "primary shock."
2. *Hematogenic (oligemic) shock*—due to a reduction in the circulating blood volume (hemorrhage, trauma, burns, etc.). This is surgical or wound shock. It is important to recognize that other factors, such as pain, heat, cold, hunger, and dehydration, may be associated, initiating or sustaining factors in shock.
3. *Cardiogenic shock*—due to cardiac failure (e.g., myocardial infarction, acute decompensation). The circulatory failure is of central, rather than of peripheral, origin. This mechanism is not often implicated in surgical shock.
4. *Vasogenic shock*—vasodilatation and loss of vasomotion due to the direct action of toxic substances on the vessel (histamine, bacterial, etc.).

It has been suggested that shock may be better understood if described in terms of the total clinical problem rather than pathophysiologic mechanisms. Such a classification would include:

1. Hemorrhagic shock
2. Wound shock
3. Burn shock
4. Toxic shock
5. Dehydration shock
6. Anesthetic shock
7. Cardiac shock

Shock is also encountered in diabetic acidosis, hypoglycemia (insulin shock), transfusion reactions, nutritional deficiencies ("chronic shock"), drug idiosyncrasy and as a complication of pregnancy.

Pathophysiology of Shock

The common denominators in shock are: an inadequate peripheral circulation and tissue hypoxia (stagnant type). Many of the immediate and delayed bodily reactions to these alterations may be analyzed in terms of hemodynamics and circulatory failure (Fig. 16).

CHAPTER 6

Shock and Hemorrhage

Wound shock is the response of the body to a complex and random event—the incurrence of a wound. Wounds exhibit a wide spectrum of tissue injury ranging from the division of a vascular trunk, to a massive crush and destruction of muscles—in addition a wound is attended by pain and fear, and under battle conditions by the rigors of transport and other exhausting stimuli The effect of a wound is that of the vector sum of its many components acting in the direction of deterioration.—CHURCHILL, 1952.

SHOCK

SHOCK HAS been described as an abnormal physiologic state which is characterized by a disparity between the circulating blood volume and the capacity of the vascular bed, resulting in tissue anoxia and circulatory failure.

The clinical entity of established shock and the need for urgent treatment must be clearly understood. The patient who has received a serious injury has often lost a large amount of blood. He is weak, faint or prostrate Although usually conscious, he is apathetic, sluggish and responds poorly. He complains of pain, thirst and coldness. He may be restless and apprehensive. The skin is pale or mottled and cold, moist and "clammy" The lips, mucous membranes and nail beds are unusually pale or cyanotic. The pulse is weak, "thready" and often rapid. The peripheral veins are collapsed and fill slowly. The blood pressure is decreased and the pulse pressure narrowed. The condition is progressive and changes rapidly. For this reason, a brief description, such as this, cannot portray the changing picture of shock. It must be seen to be understood.

Usually, several factors operate in the production of shock. The

With progressive fall in effective blood volume, the fully developed picture of shock develops. When the fall in blood volume occurs gradually, the onset of shock may be delayed; when massive hemorrhage causes a sudden fall in blood volume, shock appears immediately or in a very short time. The reduction in blood volume in mild shock usually approximates 15 or 20 per cent of the total blood volume; in severe shock, 40 per cent or more. Within the body as a whole there is a tendency toward restoration of the blood volume by the relatively slow passage of intercellular fluid into the vascular compartment. This results in hemodilution and a reduced red cell mass.

The reduction in blood volume is related directly to the severity of shock. Loss of circulating blood volume may follow external or internal hemorrhage, extravasation of blood and extracellular fluid into injured tissues, exudation from body surfaces (as in burns, or peritonitis), stagnation or sequestration of blood in the vascular system or a combination of these factors. It was previously thought that in shock a generalized loss of fluid occurred through anoxic capillary membranes, but it has now been demonstrated that such transudation occurs only into regional damaged tissues. The loss of fluid from the circulation is therefore a strictly local phenomenon and not a generalized one.

Early in shock there is a depression of circulation through the capillary bed from vasoconstriction and reduced blood volume which accounts for the clinical manifestations of skin pallor and coolness. Vasoconstriction may be sufficient to maintain systemic circulatory compensation for a short time, but it may also cause disturbances which ultimately become harmful and decrease the chances of survival. The vasoconstriction primarily involves tissues and organs not immediately essential for survival, including the kidneys. Vasoconstriction of the renal vessels and renal ischemia have been considered to be the cause of the acute renal failure which follows the prolonged hypotension of shock. It is probable that vasoconstriction of significant degree does not occur in the coronary or cerebral vessels.

Experiments have shown that certain vasoactive factors play a role. In the early (compensatory) phase of shock, the closure of precapillary sphincters causes a diversion of blood away from the capillary bed and into the venous system. In the late (decompensatory) phase, hypoactive dilatation of the terminal vascular bed results in the trapping or stagnation of blood in the capillaries and leads to a state of irreversible shock. The compensatory stage of

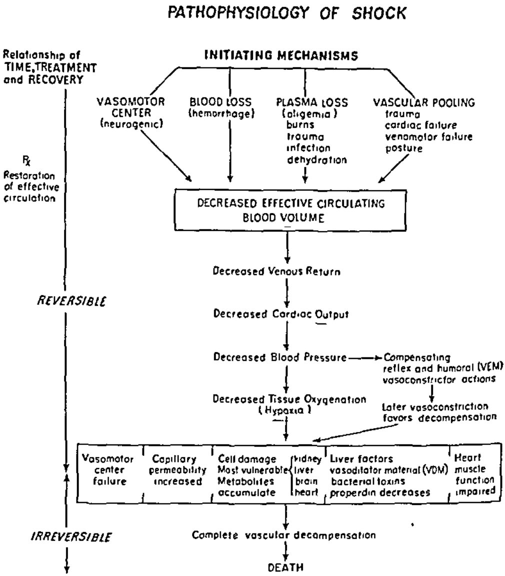

FIG 16.—Suggested by Steven Horvath, Department of Physiology, College of Medicine, State University of Iowa

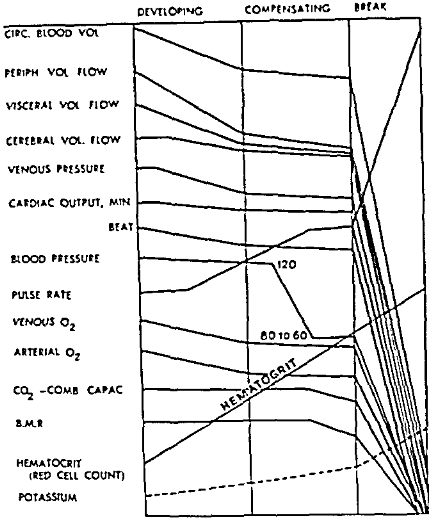

FIG. 17.—Pathophysiologic changes during development of shock. Note the early elevation of pulse rate and the relatively late fall in blood pressure. (From Cullen, S C.: *Anesthesia in General Practice* [Chicago: 4th ed., Year Book Publishers, Inc, 1954], p 258.)

pressure is measurable. As a rough guide to the decrease in total blood volume, one may say that, when the systolic pressure is below 100 mm. Hg and symptoms and signs of shock are present, the blood volume has been reduced at least 30 per cent and that, when the blood pressure is 85 mm. or below, the blood volume has been reduced at least 40 per cent or more. It is not unusual, however, for some patients to lose 25 per cent of the blood volume and not exhibit signs of shock.

Blood pressure is the result of an interplay of several factors (Fig. 17). The most important of these are the cardiac output and

shock is associated with the appearance in the blood of a vasoexcitor principle (VEM) which is formed in the anoxic kidney. At this time, a vasodilator principle (VDM) is being released from the liver, spleen and muscles, but is being inactivated by the liver. As anoxia develops, however, the liver can no longer inactivate the VDM, and it appears in the circulation in increasing amounts, producing irreversible shock. Although this concept, proposed by Shorr, Zweifach and associates, has much to support it, it has not yet been established.

Toxic substances of infectious origin may also cause shock. In the dog, for example, shock can be produced on the release of ligatures previously placed around the gastrocnemius muscles. The reaction is believed to be due to the sudden flooding of the circulation with toxins of clostridial origin produced in ischemic muscle.

It is known that the dog normally harbors intestinal bacteria, especially clostridia, in the liver and that during the shock state the organisms grow rapidly. When the dogs are protected by previous administration of aureomycin, and shock which is lethal to controls is produced, a high percentage of the treated animals survive. It appears, therefore, that in the dog infection plays a role in irreversible shock, but no conclusions can be drawn regarding the importance of these factors in man.

The venous return of blood to the right side of the heart is decreased progressively as the circulating blood volume falls. This leads to a decrease in cardiac output, a decrease in peripheral flow and a reduced blood pressure. In severe shock, stroke volume may fall from a normal of 75 ml. to 20. ml., and the cardiac output from a normal of 5,500 ml./minute to 2,400 ml./minute. There is a close correlation between the deficit in the circulating blood volume, the cardiac output and shock.

The heart rate is accelerated through carotid and aortic reflexes in an attempt to compensate for the reduced cardiac output. But the heart rate is affected by many factors and cannot be correlated as closely with the severity of shock as the blood pressure. In severe wound shock the pulse rate may actually decrease.

The blood pressure begins to fall as the compensatory mechanisms (tachycardia and vasoconstriction) fail and/or the total blood volume is depleted by about 15–20 per cent of normal. The systolic blood pressure falls relatively more than the diastolic pressure, causing a narrowing of the pulse pressure. This accounts for the difficulty in palpating the pulse in some shocked patients even when the blood

TABLE 10 —Findings in Wound Shock of Different Degrees of Severity *

Degree of Clinical Severity of Shock	Average Amount of Blood Lost (% of Normal)	Systolic Blood Pressure	Pulse Volume	Capillary Circulation of Skin	Color of Skin	Temperature of Skin	Thirst	Sensorium
No clinical signs of shock	15	Normal	Normal	Normal or slightly decreased	Normal	Normal	Normal	Clear and distressed
Slight degree of shock	20	To 20% below normal	Normal	Definite slowing	Pale	Cool	Normal	Clear and distressed
Moderately severe shock	35	Decreased 20–40% below	Decreased volume	Definite slowing	Pale	Cool	Definitely increased	Clear with some apathy until aroused
Severe shock	45	Decreased 40% or more below normal	Weak to imperceptible	Very sluggish	Ashen to gray cyanotic with mottling	Cold	Severe	Apathetic to comatose; little distress except thirst

* From Beecher, H. K., *et al*: I. The internal state of the severely wounded man on entry to the most forward hospital, Surgery 22 672-711, 1947.

the peripheral resistance. As cardiac output falls, the blood pressure falls. Early in shock, peripheral resistance is slightly increased (except in neurogenic shock), and in the decompensatory phase the peripheral resistance is progressively decreased. When hemodilution occurs as interstitial fluid shifts into the vascular compartment, decreased viscosity of the blood contributes to a decrease in peripheral resistance Generally, the function of the heart is not impaired until the later stages of severe shock. Therefore, measures designed to aid myocardial function, including digitalization, are usually ineffective.

Water and electrolyte depletion will produce peripheral vascular collapse. When injury, operation or hemorrhage is superimposed on dehydration states, the narrow balance may be upset and result in severe surgical shock. Although hypopotassemia may appear in the terminal stages of shock, it is unlikely that potassium plays an important role in the initiation or progression of the shock state.

It is probable that the adrenal glands do not play a significant role in the pathogenesis of shock except when a state of adrenal insufficiency exists.

Recognition of Impending or Established Shock States

The keynote to the effective treatment of shock is early diagnosis. It is generally easy to recognize the fully developed picture of shock, but the same cannot be said of early or compensated shock. Both the compensatory mechanisms which sustain the patient and the noxious influences which propagate it during the early phases tend to make accurate evaluation and prognostication difficult. The clinician must appreciate the fact that shock is a dynamic, not a static, process. The patient's condition is either getting worse or getting better, never standing still. The clinical picture may change rapidly, most often in the direction of deterioration. For this reason, the physician must take no chances with the patient's life when the possibility of shock exists and the signs and symptoms are compatible with early shock. He must institute appropriate therapy before the situation gets out of hand. This usually means immediate restoration of the circulating blood volume by blood transfusions.

The diagnosis of shock is made on the basis of the history and physical examination (Table 10) and a high index of suspicion in circumstances where shock is likely. The symptoms and signs in the

Laboratory Findings.—*The laboratory findings are variable, depending on the cause and duration of shock as well as on changes produced by the compensatory mechanisms.* In shock due to severe hemorrhage, there may be no immediate changes in the blood. Several hours later when hemodilution occurs, a lowered red cell count, lowered *hematocrit, lowered plasma protein, hyperglycemia, acidosis,* hypochloremia and elevated blood urea nitrogen may be noted. A polymorphonuclear leukocytosis generally appears early.

In shock without blood loss (burns, dehydration, crushing injuries, peritonitis), hemoconcentration is usual. Blood volume is decreased due to ECF loss, and blood viscosity is increased. The urine output is decreased (or absent) and of high specific gravity but usually contains no abnormal elements.

The Preventive Treatment of Shock

It is a truism that cannot be too often repeated, that *the best treatment of shock is its prevention.*

In the surgical patient, shock is best averted by recognizing and correcting, before the operation, those conditions which by summation lead to shock. Cold, fatigue, pain and anxiety may set the stage for the shock cycle. Physical and/or physiologic disturbances such as cardiovascular or pulmonary diseases, fluid, electrolyte and nutritional imbalances, anemia, hypoproteinemia or blood volume deficiency contribute to the development of shock. In general, immediate operation must be avoided in patients exhibiting signs of impending or established shock except when exsanguinating hemorrhage makes operation to save the life imperative.

The shock tendencies of the operative phase are enhanced by prolonged and deep anesthesia, extensive dissection with tissue trauma, fluid loss and exposure, blood loss (which is not replaced), body heat loss, unusual or strained positions or sudden changes in position, and impaired ventilation. Most of these factors can be controlled.

Shock during the postoperative phase commonly is due to unrecognized or continued blood loss, serious infections (especially peritonitis), hepatic or renal failure, fluid or electrolyte deficits and pulmonary embolism or coronary thrombosis. While prevention of these complications is sometimes impossible, much can be done to decrease the frequency with which they occur.

early phases will be the manifestations of reduced peripheral blood flow plus those reactions which are the result of secondary compensatory mechanisms. Later the manifestations are the result of a complete decompensation of the homeostatic mechanisms, which leads to an irreversible shock phase and death.

From a practical standpoint, irreversibility is related to time and cellular death. Local areas of cellular death can develop (necrobiosis) without death of the individual. Irreversible shock is not an end point which can be determined categorically except when the patient is dead. For this reason, every reasonable effort must be made to resuscitate the shocked patient regardless of the belief that the shock may be irreversible. Many shock states previously thought to be irreversible are now known to be reversible and will respond to energetic treatment (e g., shock due to sepsis and severe hemorrhage). This has made certain revisions in the older concepts of irreversibility necessary.

The symptoms of early shock are: weakness, faintness in the upright position, dizziness, dimness of vision, nausea, vomiting, thirst, apprehensiveness, restlessness and a sense of impending death. The principal signs are:

Lowered Blood Pressure.—Serial blood pressure determinations are most valuable in assessing the trend. If the systolic blood pressure is 100 mm. Hg or below for more than an hour in a patient in whom shock might develop, immediate steps must be taken to restore the circulating blood volume. As stated earlier, the blood pressure is often maintained for a time, then falls suddenly and dangerously when compensation fails. For this reason, blood pressure determinations alone cannot be regarded as completely reliable in detection of impending shock. The diastolic pressure falls relatively less than the systolic, and the pulse pressure is narrowed

Increased Pulse Rate.—Although variable, the rate is usually increased, and the pulse feels "soft" or of poor volume and is "thready."

Decreased Peripheral Circulation.—The extremities are cool and clammy. The skin is ashen or cyanotic. The veins of the hand may be collapsed and fill slowly when the part is lowered. The capillary bed (nail beds, lips) also refills slowly when emptied by pressure.

Impaired Cerebral Activity.—The patient may be dull, apathetic and insensitive to stimuli. As shock progresses, coma may appear. Prolonged hypotension and hypoxia sometimes lead to permanent cerebral damage.

LABORATORY FINDINGS.—The laboratory findings are variable, depending on the cause and duration of shock as well as on changes produced by the compensatory mechanisms. In shock due to severe hemorrhage, there may be no immediate changes in the blood. Several hours later when hemodilution occurs, a lowered red cell count, lowered hematocrit, lowered plasma protein, hyperglycemia, acidosis, hypochloremia and elevated blood urea nitrogen may be noted. A polymorphonuclear leukocytosis generally appears early.

In shock without blood loss (burns, dehydration, crushing injuries, peritonitis), hemoconcentration is usual. Blood volume is decreased due to ECF loss, and blood viscosity is increased. The urine output is decreased (or absent) and of high specific gravity but usually contains no abnormal elements.

THE PREVENTIVE TREATMENT OF SHOCK

It is a truism that cannot be too often repeated, that *the best treatment of shock is its prevention.*

In the surgical patient, shock is best averted by recognizing and correcting, before the operation, those conditions which by summation lead to shock. Cold, fatigue, pain and anxiety may set the stage for the shock cycle. Physical and/or physiologic disturbances such as cardiovascular or pulmonary diseases, fluid, electrolyte and nutritional imbalances, anemia, hypoproteinemia or blood volume deficiency contribute to the development of shock. In general, immediate operation must be avoided in patients exhibiting signs of impending or established shock except when exsanguinating hemorrhage makes operation to save the life imperative.

The shock tendencies of the operative phase are enhanced by prolonged and deep anesthesia, extensive dissection with tissue trauma, fluid loss and exposure, blood loss (which is not replaced), body heat loss, unusual or strained positions or sudden changes in position, and impaired ventilation. Most of these factors can be controlled.

Shock during the postoperative phase commonly is due to unrecognized or continued blood loss, serious infections (especially peritonitis), hepatic or renal failure, fluid or electrolyte deficits and pulmonary embolism or coronary thrombosis While prevention of these complications is sometimes impossible, much can be done to decrease the frequency with which they occur.

The prevention or amelioration of shock in the patient who has suffered serious injury is accomplished by attention to certain details and an appreciation of the priorities in treatment:

1. Examine the patient carefully for possible injuries beyond those which are obvious. Keep the patient under continuous observation.
2. Establish and maintain the airway, if it is obstructed.
3. In suspected, impending or established shock, institute appropriate measures (see below) immediately.
4. Immobilize fractures.
5. Protect the patient from exposure and fatigue.
6. Relieve pain and restlessness.

The Definitive Treatment of Shock

Shock is always an emergency condition. Successful treatment depends largely upon restoration of the circulating blood volume before irreversible tissue changes develop. Following injury and/or hemorrhage, there are other important items in shock treatment, but restoration of the blood volume is basic.

The essential points in treatment are:

1. Arrest hemorrhage if possible.
2. Maintain an open airway and adequate ventilation.
3. Restore the circulating blood volume.
4. Place the patient in the shock position, i.e., with the foot of the bed or stretcher elevated (about 10 in.) unless head or chest injuries contraindicate this position.
5. Keep the patient warm but do not overheat.
6. Relieve pain with opiates and anxiety with sedatives.
7. Do not begin anesthesia or operative treatment during the shock state unless required as a resuscitative measure.

Nature aids the physician in the control of hemorrhage—by means of a decrease in bleeding from fall in blood pressure, the contraction and retraction of divided vessels, the tamponade effect of hematoma formation and the increased tendency to blood clotting which follows injury. The physician can aid hemostasis by pressure, elevation of the part, ligation of vessels and use of a tourniquet. The latter rarely is necessary and, improperly used, is a dangerous device.

Generally, matched whole blood should be used for correction of the blood volume deficit. Blood for cross-matching and typing should

be obtained as soon as possible and, if necessary, plasma expanders or less effective saline or crystalloid solutions started in the interim. Saline or glucose solutions will often sustain the patient for brief periods until blood is available. If shock is due to loss of body fluid as well as blood, suitable repair solutions must be administered in addition to blood. The aim is to give the type and amount of fluid necessary to restore deficits.

Patients who have been injured and show signs of *impending shock* will usually require at least 500–1,000 ml. of blood. Patients who exhibit signs of *early shock* will require 1,000–1,500 ml. or more, and patients who show signs of *established shock* and an absent auscultatory blood pressure will require a minimum of 2,000 ml. (4 or more units) of blood rapidly. The rate and amount of blood required may be gauged by the response to transfusions. The initial 500 ml. can be given rapidly (five to ten minutes), then the second and third bottles should be given slower, or at a rate of about 500 ml. in thirty minutes. The common error is to underestimate the need for blood and to undertransfuse the patient. It is best to administer an extra 500 ml. after the patient's condition has become stabilized and signs of shock have cleared. The danger of cardiovascular overloading must be kept in mind (in the case of the very young, very old and those with cardiopulmonary injury or disease). Circulatory overloading should be suspected if there is evidence of increased venous pressure and pulmonary congestion.

The need for an open airway and good ventilation should be obvious. Hypoxia from whatever cause aggravates shock. Following thoracic injury there may be serious ventilatory impairment due to pneumothorax, hemothorax, "stove-in" chest, sucking wounds, etc. Immediate attention must be directed toward improving ventilation. In these situations, oxygen therapy will serve to decrease the adverse influence of ventilatory insufficiency; but when the ventilatory mechanism is normal, oxygen therapy is generally ineffective because tissue anoxia is the result of stagnation of the circulation (stagnant anoxia) rather than of decreased oxygen in the blood (anoxic anoxia).

The patient should be kept supine in the "shock" position. This position is conducive to improvement in the circulation to the vital medullary centers and to more efficient return of blood from the dependent portions of the body. The shock position is contraindicated, however, in patients with head or thoracic injuries. In the former, it leads to increased intracranial bleeding; and in the latter, to increased

respiratory embarrassment. Patients in whom a low level of responsiveness or coma exists should be kept in a lateral position to avoid aspiration of secretions or vomitus.

The shocked patient should be covered with blankets to conserve body heat, but external heat is generally contraindicated. Overheating is to be avoided because it produces cutaneous vasodilatation and thus deprives the vital organs of their share of an already lowered blood supply, increases local metabolism and introduces the hazard of thermal burns in patients whose sensorium is dulled by shock.

Morphine is not a corrective for shock and should not be given as a routine It is indicated for pain only and is contraindicated for patients with head or undiagnosed abdominal injuries. Frightened or apprehensive patients should be given sedatives rather than opiates. Stuporous or comatose patients need neither opiates nor sedatives, and none should be given. When these drugs are required, they are best given in small and, if necessary, repeated doses intravenously. In shock, there is, understandably, poor absorption of drugs deposited either subcutaneously or intramuscularly. When large doses of drugs are given by these routes to shocked patients, rapid absorption and drug intoxication may result on recovery.

The shocked patient should be placed at complete rest. When fractures or extensive soft-tissue injuries exist, splinting or immobilization is essential. The important considerations in this respect are described in Chapter 29, on Fractures.

The stimulants caffeine, Coramine,® camphor, etc., have no place in the modern-day shock treatment. The oral intake of coffee or warm liquids is permissible provided that injury to the gastrointestinal tract is not suspected and operation is not an immediate prospect.

In the absence of adrenal insufficiency, cortisone or ACTH are not helpful. If the patient has had a protracted illness or has been subjected to repeated stresses, the possibility of adrenal corticoid depletion must be considered, and in some instances hormone replacement therapy will be necessary.

The vasopressors also have a limited role in the treatment of shock. They may be used in special instances, e.g., in the neurogenic reaction which follows spinal anesthetic. Their administration in other types of shock, and especially traumatic shock, should be limited to those patients who have received adequate blood replacement, who are not losing blood or other fluid and who have failed to respond or are in "irreversible" shock. The drugs commonly used are Neo-

synephrine® and nor-epinephrine, which may be given by continuous-drip infusion. The dosage is regulated according to the response of the patient. In some individuals, rapid digitalization with one of the several intravenous preparations may be indicated if signs of cardiac failure appear.

HEMORRHAGE

With onset of hemorrhage, local mechanisms respond to effect hemostasis (literally "blood halt"). If blood flow is not stayed shortly, systemic reactions occur which maintain homeostasis of the organism while at the same time augmenting hemostasis locally.

The principal local hemostatic mechanisms are represented by clot formation, contraction and retraction of the damaged vessel and development of increased regional tension as a result of extravasation of blood.

With hemorrhage, blood is exposed to injured tissue, which causes liberation of thromboplastin from the tissue and blood platelets, and the clotting process begins (Fig. 18). Thromboplastin and prothrombin (plasma), in the presence of ionic calcium, interact to form thrombin. Thrombin then quickly interacts with fibrinogen (plasma) to cause precipitation of fibrin. Fibrin appears in the form of a network of long threads or strands which entrap platelets, erythrocytes and leukocytes, thus producing a blood clot. This sketchy outline of a series of complicated physicochemical reactions omits mention of various co-factors and naturally occurring countermechanisms.

As blood leaves the opened vessel, the intraluminal pressure falls. If the vessel has been completely divided, the ends will retract and contract, and the intima will curl inward. Blood flow through the vessel is diverted through neighboring channels and flow is decreased through the injured vessel. All of these changes favor platelet massing and clot formation.

Extravascular changes are the result of blood collecting in the tissues. External compression of the bleeding vessel is effected as the relative pressures inside and outside the vessel are altered. When bleeding from an artery (high pressure) occurs into the subcutaneous tissues (low tissue tension), extravasation will be great before external pressure exceeds internal (intravascular) pressure and bleeding stops. On the other hand, when bleeding occurs from a vein, the

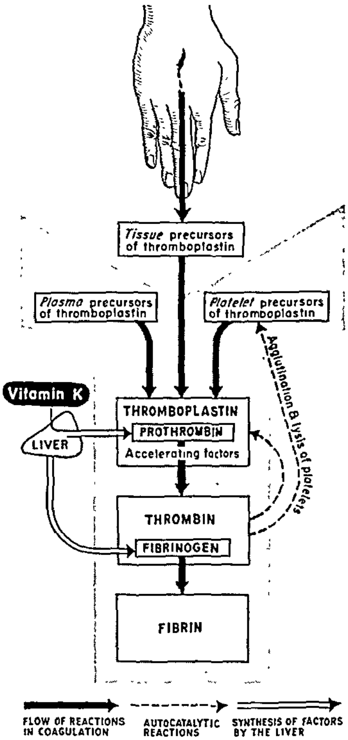

Fig. 18.—Mechanism of formation of fibrin in blood coagulation Three basic stages are illustrated: (1) *formation of thromboplastin;* (2) *conversion of prothrombin to thrombin* and (3) formation of fibrin from fibrinogen. (From "Blood Coagulation—Pfizer Spectrum," J A.M.A. Vol 152, Aug 29, 1953 [adv.])

external pressure required to stop the flow of blood will be much less because the relative tensions are more nearly equal. When bleeding occurs directly from the mouth of a divided vessel to the exterior, then tissue tension is not involved, there is no compression and bleeding will not cease until other hemostatic forces stanch the flow. The situation also obtains when the injured vessel communicates with any of the body cavities (e.g., alimentary tract, lung, bladder, etc.). If local changes about the vessel, or changes in the vessel wall (scarring, arteriosclerosis, etc.) prevent retraction and contraction, bleeding may be profuse and prolonged. On the other hand, when bleeding occurs into a closed space, counterresistance which develops as extravasation progresses will vary according to the characteristics of the compressed tissues. Thus, bleeding into a joint will cease when the joint space is filled; but bleeding into the cranium may continue and the patient may die of cerebral compression before bleeding has stopped.

Immobilization incident to injury also favors hemostasis. Pain, shock, weakness, muscle spasm and even the unconscious state may serve to decrease blood flow and blood loss.

The systemic responses to significant blood loss are similar to those of surgical shock. A reduction in the circulating blood volume and hypotension activates compensatory physiologic mechanisms. There is vasoconstriction in the nonvital areas of the body (skin, mucous membranes and splanchnic areas) which effects a reduction in the capacity of the vascular bed and a redistribution of blood so that the vital organs (brain and heart) are well supplied with oxygen. The carotid sinus and aortic arch reflexes are activated by hypotension, and the carotid and aortic body chemoreceptors are activated by anoxia, resulting in an increase in heart rate and an increase in rate and depth of respiration. The spleen contracts and an extra quota of blood is discharged into the circulation. In severe hemorrhage the clotting mechanism is accelerated and the clotting time is shortened.

The delayed response to hemorrhage is the mending process. Actually, it begins immediately after blood has been lost. First, blood volume is restored by a process of hemodilution, as fluid shifts from the tissue spaces and cells into the vascular compartment. The blood cellular and protein concentrations are thereby reduced. Protein stores of the body are mobilized to reconstitute lost plasma proteins; and later, blood cellular elements are restored through increased hemopoiesis. The rapidity of repair depends, to a large extent, on the

magnitude of the deficits and the nutritional state and dietary intake of the patient.

Symptoms and Signs

Hemorrhage may result from arterial, venous or capillary injury. Arterial bleeding is brisk, spurting and pulsatile; the blood is bright red Venous bleeding is less vigorous, under lower pressure and non-pulsatile; the blood is dark red. Capillary hemorrhage is manifested by a continuous oozing.

Blood loss may be of minor, major or massive proportions. In the healthy person, sudden losses in the range of 700 ml. (about 14 per cent of total blood volume) usually produce no serious sequelae. Losses up to about 1,500 ml., or 30 per cent of the total blood volume, are dangerous to life but may usually be tolerated without transfusions. Losses in excess of 1,500 ml. are likely to be fatal unless transfusions are given.

Blood volume is proportional to total body water and lean-body mass. The total body water varies directly with the lean-body mass and indirectly with the body fat. Fat stores contain little water and are quite inactive metabolically. The very lean person in health has relatively more total body fluid (and total blood volume) than the very obese person. The healthy person of average obesity has about 85 ml. of blood per kilogram of body weight. In the very obese person, the figure may be 60 ml./kg. and in the very lean person 100 ml./kg.

Estimated blood loss of 30 per cent in a 60 kg. person of average obesity would be:

$$60 \text{ kg.} \times 85 \text{ ml.} \times 30\% = 1{,}530 \text{ ml. blood loss}$$

Bleeding may be external or internal; i.e., blood may appear on the surface of the body or blood may accumulate within the body. In external hemorrhage the source of bleeding and its magnitude is often apparent. Internal hemorrhage may be difficult to recognize, localize and quantitate. Internal hemorrhage after trauma or operation may often be suspected, but spontaneous and concealed hemorrhage is a different matter and may not be suspected until signs of severe blood loss develop (e.g., ruptured ectopic pregnancy).

The complications which may result from hemorrhage and treatment of hemorrhage are numerous. Injury to important blood vessels, either from the original trauma or incidental to attempts to control blood loss, may compromise the peripheral circulation and the viabil-

ity of distal structures (e.g., laceration of the popliteal artery and/or prolonged application of a tourniquet usually leads to gangrene of the leg). Pressure from an enlarging hematoma may produce vessel and nerve damage; or in the chest, may lead to decreased pulmonary function, cardiac tamponade, etc. Hematomas, per se, predispose to infection, soft-tissue calcification or ossification. Blood transfusion itself may lead to complications more serious than those resulting from blood loss.

The signs of hemorrhage, besides the appearance of gross or changed blood, are:

1. Restlessness and apprehension
2. Pallor of the skin and mucous membranes
3. Cold moist skin
4. Thirst
5. Deep sighing respirations (air hunger)
6. Full bounding pulse, early, then "thready" pulse
7. Tachycardia which may be unexplained (a valuable sign of concealed hemorrhage)
8. Delayed signs: hypotension, decrease in hemoglobin, red cell and hematocrit values and the full-blown picture of shock

Treatment

Bleeding of minor degree usually ceases spontaneously if the hemostatic mechanisms are normal. The normal response is facilitated by elevation, external compression, alteration of local temperature (cold or heat), rest and time.

Active bleeding requires active treatment. The local measures available for staying hemorrhage include: ligation, cauterization, tamponade (packing), application of vasoconstrictors (usually epinephrine) and introduction of hemostatic substances. The hemostatic substances available are: thrombin, fibrin, fibrinogen, thromboplastin, gelatin sponges and oxidized cellulose. Tourniquets should be used only in case of emergency and should never be left in place longer than thirty minutes without releasing them. They may be reapplied if needed.

All direct measures, including ligation, should be carried out under aseptic conditions, if possible. The chances of a complicating wound infection are multiplied when this rule is violated. However, if exsanguinating hemorrhage exists, one cannot delay treatment, and

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All direct measures, including ligation, should be carried out under aseptic conditions, if possible. The chances of a complicating wound infection are multiplied when this rule is violated. However, if exsanguinating hemorrhage exists, one cannot delay treatment, and

"clean surgical care," rather than aseptic care, must be acceptable. The primary consideration must always be to save life.

After hemorrhage has been controlled, or concurrent with attempts at control, appropriate measures must be instituted to avert or treat shock. In the presence of internal hemorrhage it often is advisable to reverse this order; that is, first the circulatory deficit is corrected by transfusion, then direct control of bleeding is achieved by operation (e.g., bleeding peptic ulcer). The timing of the operation obviously is of critical importance.

Blood Transfusion

Blood transfusion is indicated if blood loss has been sufficient to produce a reduction in the effective circulating blood volume. Whole blood must be given promptly and in amounts sufficient to correct the physiologic deficit. Therapeutically, with the exception of arrest of hemorrhage and maintenance of respiration, blood replacement has highest priority.

There are no satisfactory substitutes for blood, although several "blood substitutes" are available. None of the substitutes contain elements of the same particle size or specialized functions of the red blood cell. Volume for volume, blood produces a more lasting increase in the intravascular mass because it is not exchanged in other compartments.

The amount of blood needed can be judged on the basis of the history of blood loss, evidence of gross bleeding, size of the wound, general appearance of the patient and the blood pressure. In the period immediately following hemorrhage there is little to be gained from laboratory studies

After severe hemorrhage the approximate blood requirements of an average patient are:

Impending shock—marginal systolic pressure	500–1,000 ml
Established signs of mild shock—systolic pressure 90 mm Hg or below	1,000–1,500 ml or more
Established severe shock—systolic pressure unobtainable	2,000 ml. or more

Blood should be given rapidly at first (500 ml. in thirty minutes or less) in order to bring the systolic pressure up to low normal (about 100 mm. Hg). As soon as this has been accomplished, the rate may be decreased. Although complete blood replacement is not immediately

necessary, sufficient blood must be given to effect a good clinical response. If the response is unsatisfactory, the reason or reasons for failure must be determined and corrected.

1. Is there concealed continued blood loss or an overlooked cause for shock?
2. Has blood replacement been inadequate? Blood losses generally are underestimated.
3. Has too much blood been given? The patient with marginal cardiac compensation is "overloaded" easily.
4. Is there primary cardiac failure on the basis of pre-existing myocardial disease?
5. Is there failure of peripheral resistance? Loss of vasomotor tone occurs in advanced ("irreversible") shock.

Serious transfusion reactions occur despite special precautions to avert them. Many such reactions are due to human errors in labeling, cross-matching, interpretation, administration, etc., which are difficult to control; but others are easily avoided.

1. Do not use Group IV blood ("universal donor") unless it has been cross-matched with the patient's blood.
2. Do not transfuse an Rh negative patient with Rh positive blood, except in an emergency.
3. Administer the first 50–100 ml. of blood slowly (if possible) and watch for any immediate reaction. If a reaction occurs, discontinue the transfusion. Serious complications are unlikely from a small amount of blood.
4. *Be sure that the patient receives the right blood.* This statement may appear superfluous, but experience has taught otherwise. Patients have died from understandable, but usually inexcusable, errors. The physician must exercise constant vigilance to see that no harm comes to his patient.

Transfusion Reactions

Pyrogen.—Chills and fever appear shortly after the transfusion is begun They are never fatal. They often may be avoided by flushing the intravenous tubing with pyrogen-free saline solution before starting the transfusion. Calcium gluconate (10 ml. of a 10 per cent solution IV) and, if necessary, an opiate will help.

Urticarial.—Hives or angioneurotic edema may develop. Administration of epinephrine, calcium gluconate and antihistaminics is

indicated according to the severity of the reaction. If edema of the glottis develops, tracheotomy must be considered.

CIRCULATORY OVERLOAD.—Probably the most common cause of death from infusion or transfusion is circulatory overload. The reaction may be countered by applying tourniquets to the extremities to decrease venous return to the heart (both a diagnostic and first-aid measure), oxygen therapy and venesection.

HEMOLYTIC.—Chills, fever and severe pains in the back, flanks and substernal region occur soon after transfusion is begun. The urine will contain hemoglobin, and oliguria or anuria will develop Jaundice may appear in about twelve hours. Therapy includes stopping the transfusion, treatment of shock with properly matched blood and management of the acute renal insufficiency if it develops.

SERUM SICKNESS.—Fever, lymphadenopathy with splenomegaly, backache, joint pain and stiffness may appear a week or two after transfusion. The reaction is always delayed and never fatal. Calcium gluconate (intravenously), antihistaminics and cortisone are beneficial.

BLOOD DERIVATIVES AND PLASMA EXPANDERS (BLOOD SUBSTITUTES)

Blood plasma, lyophilized plasma and human serum albumin are used to effect an increase in the total blood volume and to restore osmotic tension They do not directly influence the oxygen-carrying capacity of the blood. The protein of these blood derivatives is not used for protein synthesis, and for this reason they have little place in the treatment of malnutrition. Fresh plasma or frozen plasma prepared from fresh blood contains prothrombin, complement and antihemophiliac globulin Stored plasma and plasma prepared from outdated blood are low in these substances. A most serious objection to pooled plasma, which was used liberally during World War II, is the high incidence of homologous serum jaundice. Since then, the incidence of this complication has decreased, chiefly as a result of more careful selection of donors and special processing of the plasma. It is still advisable to reserve pooled plasma for emergency use only.

Concentrated salt-poor human serum albumin is an effective substitute for pooled plasma. It may be used in acute oligemic shock when whole blood is not available. It is a valuable physiologic hyperosmotic solution for the reduction of edema (pulmonary edema, cere-

bral edema) and is sometimes given for the rapid correction of nutritional hypoalbuminemia. Serum albumin is expensive, and its action is of short duration.

Plasma expanders are substances which, when infused into a patient in shock, will temporarily increase the blood volume. They may be used to tide the patient over until more appropriate replacement therapy can be given or until the patient is able, through normal responses, to restore the depleted blood volume. The importance of these substances in the event of total warfare is obvious, and for several years they have been stockpiled by the government. In civilian practice they constitute an important item in the physician's emergency kit, in the accident room, in the first-aid station and also in the operating room. These preparations can be easily stored and quickly administered should an emergency arise.

Many plasma expanders have been investigated, but only a few have proved acceptable. Of these, dextran, polyvinylpyrrolidone (PVP) and gelatin are most promising.

Dextran is a polysaccharide produced by bacterial fermentation of sucrose. It is made up in 6 per cent concentration in saline solution and has approximately the viscosity of plasma. It is an efficient plasma expander. Dextran is excreted mainly through the urine. There are no serious side reactions to its use, but difficulties in blood typing have occasionally been noted. For this reason, if blood is to be given following dextran, a sample of the patient's blood should be obtained for typing and cross-matching before starting dextran.

Polyvinylpyrrolidone is a synthetic water-soluble substance made from a mixture of acetylene, ammonia and formaldehyde. It was first used by the German Army in World War II. It effectively increases and maintains the blood volume of shocked patients. About 50 per cent is excreted in the urine during the first day, and smaller amounts for an indefinite period. It is possible that PVP is stored in the tissues, especially the liver and spleen, for long periods. Whether this is harmful has not been established.

Gelatin was first used in the treatment of shock forty years ago. Despite many improvements in the preparation of this substance, it is still not an entirely satisfactory plasma expander. One serious disadvantage of the usual gelatin solution has been that it is a gel at room temperature. Certain modified forms (oxypolygelatin, etc.) appear more promising.

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CHAPTER 7

Surgical Infections and Their Treatment

SURGICAL INFECTIONS

SURGICAL INFECTIONS encompass a broad span of conditions that are due to micro-organisms. They may result from a single species (monomicrobic infection) or from a mixed bacterial population (polymicrobic infection). The infection may be acute, subacute or chronic, according to the offending organism, the response of the host tissues and the clinical course. Most surgical infections are characterized by a local inflammatory response with destruction of tissue. The infection may take one of two clinical forms: *invasive infection*, which results from growth and spreading of organisms into the tissues, where they produce local and often systemic effects; and *noninvasive infection* (wound suppuration), in which case there is surface growth of bacteria without extension beyond the limits of the wound.

The local breakdown of tissue and the formation of an inflammatory exudate may lead to an abscess and an accumulation of pus. Recovery usually follows resorption of the exudate or external drainage, either spontaneous or surgical. The scarring produced depends upon the intensity of the inflammatory reaction and the extent of tissue destruction. Surgical infection usually follows a break in the continuity of the covering tissues, which permits entrance of pathogenic organisms. Diseases such as diabetes, arteriosclerosis and malnutrition often impair the natural defenses to infection and hence increase somewhat the virulence of the pathogen.

In contrast with surgical infections, medical infections are usually

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granulating wounds, such as those resulting from burns. Its presence is denoted by the characteristic blue-green pus which it evokes.

A mixed bacterial flora may result in invasive or noninvasive infection according to the predominant organisms, the local conditions in the wound and the response of the host. Wounds originally contaminated with a single pathogen are often cross-contaminated as a result of careless or improper handling. The need for asepsis and clean surgical care ("aseptic conscience") in wound management should be obvious.

Occasionally in polymicrobic infections the virulence of one or more of the organisms present in the wound is enhanced as a result of bacterial synergism. For example, in postoperative progressive bacterial synergistic gangrene, the combined effects of nonhemolytic micro-aerophilic streptococci and staphylococci results in an obstinate, spreading gangrene which cannot be produced by either pathogen alone

Although, ideally, a precise bacteriologic diagnosis is desirable in every infection, this is not always necessary or practicable. Often such studies are time consuming and costly; and the information required for treatment can be derived from observing the local reaction, the type of exudate and studying a simple Gram-stained smear of the pus. When the infection promises to be severe, protracted or resistant to treatment, both aerobic and anaerobic cultures should be made, the organisms should be identified and in vitro sensitivity tests run to determine the most effective antibiotic agent or agents.

Response to Infection

The phenomenon of tissue reaction to injury which is called *inflammation* (Fig. 19) may be induced by pathogenic bacteria or may result from physical, chemical or ischemic injury. If it develops in the absence of bacteria, it is called a *sterile inflammation*. If it is produced by bacteria, it is called a *septic inflammation*. The local and systemic reaction to injury is essentially similar, regardless of the nature of the agency evoking the reaction. In surgical infections, the changes evoked locally by the invasion or surface growth of microorganisms require the most attention.

Inflammation is a purposeful reaction designed to localize, destroy and remove the irritant agent. The gross changes in the tissue induced by inflammation described centuries ago by Celsus consist of heat,

monomicrobic, produce little or no tissue necrosis and excite a maximal systemic response, including an immune reaction. They are generally not localized and not amenable to surgical measures.

The organisms most commonly encountered in surgical infections include.

1. *Pyogenic invasive organisms*

   Staphylococci, especially Micrococcus pyogenes. This common offender is capable of producing infection in all tissues of the body. It is a pathogen largely responsible for furuncle, carbuncle, paronychia, felon, osteomyelitis and a host of other conditions. The exudate produced is characteristically thick and creamy and has a yellow or off-white color.

   Streptococci (hemolytic, nonhemolytic, aerobic, micro-aerophilic anaerobic). Local infections, which usually result from the Group A hemolytic strains, are common in the upper respiratory passages and are easily transmitted to wounds by careless handling. These infections may take a variety of clinical forms, including cellulitis, gangrene, lymphangitis, abscess, bacteremia and septicemia. The exudate is generally thin, watery and yellowish or slightly green in color.

2. *Less common pyogenic organisms*

   Diplococci (Pneumoniae and Hemophilus influenzae). These organisms are infrequently found in pure culture. They may produce pneumonitis or pneumonia, and sometimes they invade the pleura or peritoneum.

3. *Anaerobic spore-forming organisms*

   The clostridial infections include. tetanus (Clostridium tetani), clostridial myositis and anaerobic cellulitis. Clostridium perfringens (welchii) is the most common of the pathogens producing clostridial myositis and anaerobic cellulitis. Less frequent offenders include C. novyi, C septicum, C. bifermentans (sordelli), C. sporogenes, and C. histolyticum. Growth and extension of these organisms is aided by anaerobic conditions (reduced oxygen tension), foreign bodies, dirt, necrotic tissue and ischemia.

4 *Organisms resident in the gastrointestinal tract*

   Enterococci (streptococcus) is the predominant pathogen, but a combination of organisms is encountered These may include Escherichia coli, Aerobacter aerogenes, Pseudomonas aeruginosa, Proteus vulgaris, Klebsiella pneumoniae, Alcaligenes faecalis and the paracolon bacilli Staphylococci and strains of streptococci may also appear.

Escherichia coli frequently appears in mixed infections, especially of the urinary tract. Pseudomonas aeruginosa has a low pathogenicity but a high nuisance value and is a common contaminant of

granulating wounds, such as those resulting from burns. Its presence is denoted by the characteristic blue-green pus which it evokes.

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pain, swelling and redness (calor, dolor, tumor and rubor). John Hunter added a fifth sign, impaired function.

Changes which follow the introduction of irritant substances (bacterial or otherwise) into the tissues involve vascular, cellular, humoral, neural and chemical responses. First there is injury to the cellular membrane, which results in fluid leaking out of the injured cells and out of the damaged capillaries into the intercellular space. The fluid which escapes into the tissues is called an *exudate*. It will vary in quantity, consistency and composition according to the incit-

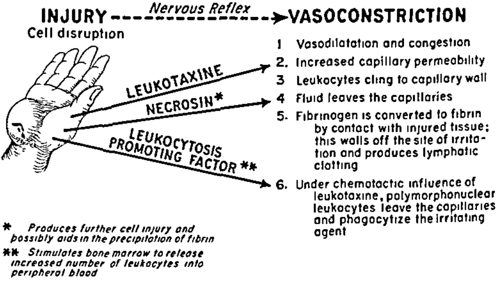

FIG 19 —Local response in inflammation. (After Menkin. Redrawn from Scope, no 4, July, 1943, p 3, courtesy of the Upjohn Company.)

ing agent, the tissue reaction and the time interval. If it is composed primarily of fluid from injured cells and blood and is relatively low in admixed cells, it is called a *serous* exudate. If it contains many erythrocytes, it is *hemorrhagic;* if it is filled with dead and disintegrating leukocytes, it is *purulent,* and if it contains a high admixture of fibrin in a purulent exudate, it is *fibrinopurulent.*

In inflammation there always is a disturbance in the normal mechanisms of fluid exchange The pressure gradient going from artery to vein increases, resulting in progressive loss of protein along the course of the vessel. The permeability gradient also increases, as demonstrated by the greater tendency of dyes to penetrate the capillary endothelium at the venous end as the vascular gradient increases. The increased capillary permeability was originally considered

due to liberation of histamine, but Menkin has ascribed it to a substance called "leukotaxine." This substance also has to do with diapedesis of leukocytes through the capillary wall.

Normally, leukocytes flow in the axial stream—that is, in the center of the capillary stream. Immediately after an inflammatory insult or stimulus, there is a very transitory vascular contraction, followed by dilatation, which persists. Capillary pressure increases. The leukocytes fall out of the stream and marginate at the border, which is known as the "zonal layer." They then make their way between endothelial cells into the tissues toward the site of inflammation. This whole process constitutes an example of chemotaxis.

The cellular response to inflammation first appears to involve the polymorphonuclear leukocytes. The polymorphonuclear leukocytes contain an intracellular enzyme (leukoprotease) which has the property of aiding the digestion of ingested particles. A number of other enzymes have also been described. As inflammation progresses, the pH locally tends to fall. This change in reaction is probably determined to a significant degree by the local presence of anaerobic glycolysis, with the conversion of sugar to lactic acid. As the pH falls, all living leukocytes disappear and only pus is seen.

The local changes evoked by bacterial invasion tend to limit the process In part, this localization or fixation is mechanical in nature. The lymphatics are blocked by plugs of fibrin or lymphatic thrombi. This barrier may be preserved if there is a balance between factors which tend to enhance the deposition of fibrin and factors which tend to inhibit or destroy it Some strains of streptococci liberate fibrinolysins, which destroy the natural barrier. Other organisms, such as staphylococci, liberate an enzyme, coagulase, which enhances the clotting tendency.

Foreign proteins may be held in the inflamed area, and antibodies (globulins) presumably aid in the direct attack on bacteria at the site of inflammation.

To a degree, the greater the local reaction, the less the tendency to bacterial invasion However, severe local reactions lead to local tissue destruction Thus, if the natural defenses of the body alone are allowed to combat the infection, a price must be paid for the fixation of the offending organisms in the tissues.

These aspects of local fixation are best exemplified by two common types of surgical infection. A streptococcus infection causes relatively little local necrosis. There is a scant fibrin barrier and a late

and ineffective lymphatic blockade. Enzymes such as streptokinase and streptodornase may destroy the barrier as fast as it forms. Spreading factors, such as hyaluronidase, may produce rapid intercellular infiltration of infectious exudate. A staphylococcus infection, on the other hand, produces a severe local reaction with early and effective lymphatic and fibrin blockade. A local highly cytotoxic substance called "leukocidin" is elaborated. The area becomes walled off and tissue invasion is arrested, but at the cost of tissue death and abscess formation

The phagocytosis-inducing substances, opsonins and tropins, are present in normal and immune sera. These are deposited as a film

TABLE 11.—STAGES OF INFECTION (AFTER BERMAN)

PATHOLOGIC CHANGES	SYMPTOMS AND SIGNS
Hyperemia	Heat and redness
Exudation	Swelling
Stasis (thrombosis of capillary and lymphatic channels)	Beginning localization
Necrosis from toxins and pressure	Pain and tenderness
Suppuration	Pus and fluctuation
Immunity (formation of the pyogenic membrane)	Hard area or wall around the inflamed zone
Granulation	Proud flesh if excessive
Epithelization	New skin
Cicatrization	Scar formation

over the material to be ingested by phagocytes, thus facilitating the process of phagocytosis. In general, the efficacy of phagocytosis hinges on their concentration.

There is also a factor, which is believed to act on the bone marrow by way of the blood stream, which has the property of inducing a discharge of immature leukocytes. This is the leukocyte-producing factor (LPF of Menkin), which is a by-product of injured cells, in inflammatory exudate. Other factors which have been demonstrated in the exudate include: necrosin, a cytotoxic substance, pyrexin, a fever-inducing substance, and leukopenin, a leukopenia-inducing substance.

If the walling-off process is imperfect or invasion is rapid, cellulitis, lymphangitis, phlebitis or septicemia may develop. When tissue breakdown occurs, an abscess or ulcer forms. In deeper tissues an abscess usually results, in superficial tissues, rupture of the abscess may cause an ulcer. A simple abscess, such as a furuncle, is made up of a central necrotic core containing bacteria, leukocytes, fibrin and

cellular debris in various stages of disintegration. Increasing tissue tension together with the catalytic action of the leukocytes is responsible for thinning and ultimate necrosis of the superficial layers of the skin. This results in the development of a pus blister, which often ruptures spontaneously, allowing the abscess to drain. (See Table 11 for the stages of infection.)

Considerations in Treatment

The major factors which condition the severity of a surgical infection are: (1) the type, virulence and dosage of the pathogenic organisms which gain entry to the tissues; (2) the time interval from inoculation to the beginning of treatment; (3) local resistance of the tissues; and (4) the general resistance of the patient.

The major considerations in the treatment of surgical infections are to:

1. Minimize the bacterial inoculation of tissues
2. Begin treatment as early as possible
3. Improve the local situation in the wound by all available means
4. Utilize specific antibacterial therapy
5. Support and maintain normal metabolism and other body functions

Common Surgical Infections

Furuncle.—A boil (Fig. 20) is a common surgical infection. It is caused most often by Staphylococcus aureus, which reaches the deeper areas of the skin through the hair follicles or sebaceous glands. The inflammatory reaction is intense, leading to tissue necrosis and the formation of a central core. This is surrounded by a granulation tissue "pyogenic membrane" and a peripheral zone of cellulitis. The infectious process is usually self-limited, but it may be complicated by inoculation of the adjacent skin and the formation of "satellite" abscesses. The usual principles of surgical treatment, including antibiotics, apply in the management of this condition. Incision and drainage is not often required if treatment is begun early. Diabetes mellitus is a frequent predisposing cause of furunculosis.

Carbuncle.—A carbuncle (Fig. 21) is a multi-loculated suppurative inflammatory process of the skin and subcutaneous tissues caused by the Staph. aureus. The pathogenesis of the infection is identical

to that of the furuncle. Since the introduction of specific antibacterial therapy, radical surgical procedures have little place in the management of carbuncles and are seldom indicated.

WOUND INFECTIONS.—Any wound is subject to bacterial contamination. Prophylactic measures that minimize the bacterial contamination of wounds greatly simplify the problems of treatment. The sources

FIG 20.—Furuncle of the "dangerous area of the face." The area is bounded by the bridge of the nose and the angles of the mouth Infections in this location should not be disturbed by incision, squeezing or other types of manipulation.

of contamination include: the patient's skin, clothes, soil, contaminated dressings, foreign bodies, the hands of attendants and the expired air of the patient and his attendants. Wound contamination by organisms of the upper respiratory tract is a special hazard when a large surface area is exposed (e.g., burn). Many sources of wound contamination exist in the operating room, but most of these can be controlled by proper aseptic technic.

CELLULITIS.—Cellulitis is an acute inflammatory reaction which extends along connective tissue planes and across intercellular spaces.

There is widespread swelling and redness, without definite localization and with little or no pus formation. An area of cellulitis appears at the periphery of most of the pyogenic infections, but the classic picture of a spreading cellulitis is the result of invasive streptococci. The zone of infection is fiery red, hot, swollen, painful and extremely tender. There may be no definite line of demarcation. Blebs and bullae sometimes form on the skin. Later there may be extensive necrosis with abscess formation. Nonoperative treatment is indicated

FIG. 21.—Severe carbuncle of the neck in an elderly obese patient with diabetes mellitus. Such extensive infections are now uncommon. Patient recovered following control of the infection, excision of the necrotic tissue and skin grafting.

unless localization occurs, at which time drainage may be required.

LYMPHANGITIS.—This condition is characterized by the presence of red streaks, which course centrally from the area of acute infection, associated with swelling and tenderness of the regional lymph nodes. A lymphangitis is secondary to a focus of infection and results from the passage of bacteria and other irritant materials into lymphatic channels. It represents a normal defense reaction against invasive bacterial infection. Surgical incision is harmful. Resolution of the process is hastened by antibiotic therapy and use of methods that decrease edema, improve the local conditions and aid the lymphatic and venous drainage.

PHLEBITIS.—Pathogenic bacteria which gain entrance into a vein induce an intense inflammatory reaction which leads to thrombosis, often with marked disturbance of the distal circulation. Thrombophlebitis may also be of traumatic or chemical origin. The process may lead to suppuration, but this is a rare complication. Thromboembolic accidents do not commonly result from thrombophlebitis of infectious origin. Thrombophlebitis must be differentiated from phlebothrombosis, or spontaneous intravascular clotting without inflammation, in which the danger of pulmonary embolism is great.

TETANUS

Clostridium tetani is a normal inhabitant of the intestinal tract of the various herbivora and, less frequently, of man. It is saprophytic in nature and noninvasive. The organism becomes pathogenic when it is introduced into wounds (especially puncture wounds), where nonviable tissue, anaerobic conditions and pyogenic infection favor its development. The incubation period for the disease varies but is usually five to fourteen days. Generally, the shorter the incubation period, the more serious the prognosis. Spore forms of the organism often remain in tissues for months or years before they become active and germinate as a result of trauma or other manipulation.

The clinical effects of tetanus infection result from one or more of the following types of involvements: (1) toxins acting locally on the effector cell (skeletal muscle) to cause "local tetanus" and (2) passage of the toxin into lymphatic channels and the systemic circulation, then to specifically reactive cells of the spinal cord and the medulla.

SYMPTOMS AND SIGNS.—The wound may be trivial and its appearance be of little aid in diagnosis. Tetanus is characterized by stiffness of the muscles, locally or generally, spasm of the pharyngeal muscles, trismus (lockjaw) and risus sardonicus, tonic and clonic convulsions.

PREVENTIVE TREATMENT.—Extensive tissue damage, gross contamination and anaerobic conditions in the wound provide a fertile soil for clostridial infection. Proper local treatment consists of surgical débridement, wound irrigation and leaving the wound opened widely. If the patient has not been actively immunized, he should be given 3,000 units of tetanus antitoxin (minimum dose) after intracutaneous and conjunctival sensitivity tests. If the patient is sensitive, he must

be desensitized.* The tetanus antitoxin should be repeated in about seven days, especially if there is to be remanipulation of the wound. If the patient has been actively immunized within five years, 1.0 ml. of the toxoid should be administered as a booster dose. But if the wound involves the head or neck (with possibly a short incubation period), both tetanus toxoid and antitoxin should be given regardless of when the patient last received artificial active immunization. Remember that it takes time to "recall" active immunity with toxoid. Patients who have never received toxoid should be given the first dose at the time they are given tetanus antitoxin. The injections required to evoke active immunity are then completed later. If tetanus toxoid and antitoxin are given simultaneously, they should be given in separate syringes and in different areas.

ACTIVE TREATMENT.—The mortality is high (about 50 per cent), but much can be accomplished by treatment. The following routine is suggested:

1. Isolate the patient in a quiet, dimmed room and start treatment immediately.
2. Administer 50,000 units or more of antitoxin by intravenous or intramuscular routes. Infiltrate 10,000 units or more around the periphery of the wound preparatory to wound excision one hour later. Begin toxoid immunization.
3. Obtain direct smears and aerobic and anaerobic cultures in an effort to establish a bacteriologic diagnosis.
4. Excise or completely expose the wound, if possible. The wound may be covered with an activated zinc peroxide suspension dressing.
5. Additional antitoxin may be given each day, 50,000 units or more.
6. Administer doses of penicillin and broad-spectrum antibiotics.
7. Use Avertin,® 60–80 mg./kg. of body weight, or paraldehyde in oil by rectum, 10–40 cc., to control spasms and convulsive

* Suggested desensitization schedule:

TIME	DOSE	DILUTION
30 minute intervals	0.1 ml.	1:10
	0.2	
	0.5	
	1.0	
	2.0	
	0.5 ml.	Undiluted
	1.0	

Continue until total dose has been given.

seizures. Curare or curariform drugs are sometimes indicated.

8. Maintain an adequate airway and ventilation. Tracheostomy is often indicated. Support of respiration by mechanical means may be necessary. Survival depends on respiration.
9. Give attention to the fluid intake and nutrition, oral hygiene, bladder and bowel function.
10. Special nursing care and constant medical supervision are essential in severe tetanus. Without them the patient dies.

Gas Gangrene

The clostridia of gas gangrene are natural inhabitants of the soil and of the intestinal tract of man and most herbivorous animals. The most common pathogen is C. perfringens (welchii). Less frequently, C. novyi, C. septicum, C. bifermentans and C. histolyticum are found. An active gas infection occurs if the clostridia are inoculated in large number into wounds containing crushed (or devitalized) tissue, dirt, other organisms and in which anaerobic conditions exist. This set of circumstances obtains in (1) accidental wounds, especially compound fractures, (2) gunshot wounds and battle injuries, (3) contused wounds and puncture wounds which have been soiled by fecal material and (4) wounds that have been closed by primary suture after inadequate preparation. The lower extremities and the buttocks are the sites most commonly involved. Because the organisms of gas gangrene are widespread in nature, it is surprising that this infection does not develop more frequently.

There are two clinical forms of the infection. *Clostridial myositis*, of the spreading or diffuse type (true gas gangrene), is an infection of the muscles which is serious and often fatal. *Clostridial cellulitis* is usually limited to subcutaneous tissue and fascial planes. The muscles are not primarily involved. This is also a serious infection but it does not have the lethal potentialities of clostridial myositis

Symptoms and Signs.—The onset of gas gangrene may occur within a few hours or from three or four days. Pain in the wound is out of proportion to the extent of the injury. The pulse rate increases, and the temperature rises The patient is acutely ill, but he may say that he never felt better. There is a spreading area of swelling about the wound, and a thin, dirty-brown exudate containing gas bubbles appears from it The drainage has a characteristic smutty odor. Crepitation in the tissues denotes the presence of gas but is not pathog-

nomonic of clostridial infection. Before other clinical signs are present, x-rays (Fig. 22) may reveal gas in the subcutaneous tissues, which extends along fascial planes. Marked swelling develops, and a characteristic dusky or bronze discoloration of the skin appears. Gangrene finally occurs. The muscle groups involved can be recognized by their gross appearance. They appear cooked and do not contract or bleed.

Direct smears of the exudate will reveal gram-positive bacilli.

FIG. 22.—Open-comminuted fracture of both bones of the forearm, complicated by gas bacillus infection (clostridial myositis). Note presence of large collections of gas in soft tissues

Deep-meat (beef heart) infusion broth is used for anaerobic culture. So-called "stormy fermentation" of milk under reduced oxygen tension is a cultural characteristic of C. welchii.

PREVENTIVE TREATMENT.—Wounds that are grossly contaminated should receive meticulous wound toilet and surgical débridement and should be *left open*. Activated zinc peroxide suspension placed in the wound is recommended as a prophylactic and therapeutic measure. Aqueous penicillin in massive doses is of value. Polyvalent antigas gangrene serum, 40,000 units or more, may be given by the intramuscular route.

ACTIVE TREATMENT.

1. Isolate the patient and use strict, aseptic precautions.

2. Provide aerobic conditions and relieve tension throughout the wound. Open it widely and remove all nonviable tissue by sharp dissection This may require excision of entire muscle groups.
3. *Amputation of an extremity may become necessary.* An open-type amputation only should be done.
4. The local use of activated zinc peroxide suspension is of value.
5. Massive-dosage penicillin or broad-spectrum antibiotic therapy should be instituted.
6. Antigas gangrene serum is of little value once the infection is established. X-radiation therapy has been all but abandoned.
7. The rapidly developing anemia (hemolysis of red blood cells) requires multiple transfusions.
8. General supportive treatment is of great importance.

Infections of the Hand

The human hand is a complex structure composed of delicately balanced tendons, muscles, joints, bones and nerves. These tissues are functionally co-ordinated, and damage to any area may result in widespread loss of function. Serious infections may jeopardize the extremity or life itself. Too often, poor management results in a crippled, useless hand.

A knowledge of the surgical anatomy of the hand and of the possible pathways of extension of infection is fundamental to intelligent treatment. Repeated, careful examinations of the hand may be necessary before diagnosis can be made. Premature incision before localization has occurred may be damaging. The requirements for rational and effective treatment of hand infections are:

1. An exact diagnosis and a precise knowledge of sites of localization and the routes of spread
2. An appreciation of the value of immobilization (Fig. 23), local heat and elevation in effecting localization
3. Specific antibacterial therapy based on a bacteriologic diagnosis if possible
4. Intelligent and timely use of surgical drainage for localized infections
5. *Avoidance of wound contamination and mixed infections*
6. Restoration of function as the ultimate goal of therapy

Minor Hand Infections.—The minor infections of the hand include felon, paronychia, furuncle, carbuncle and subepithelial abscess.

Felon.—The pulp of the finger-tip is composed of fat enclosed in a tough radiating network of connective tissue which forms a potentially closed space. Through a puncture wound (wire, nail, bristle, etc.), bacteria gain entry to this area and cause rapid swelling

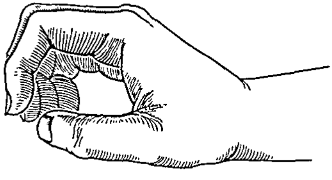

FIG 23.—The "position of function" of the hand. The hand always should be immobilized in this position if possible. If a tennis ball is held in the palm and the wrist is slightly extended, the fingers, thumb and wrist assume the proper position.

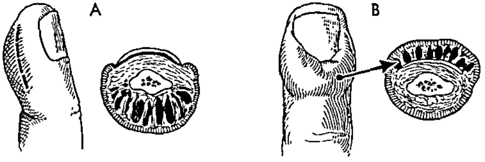

FIG. 24.—Common finger infections. A, a felon, an infection involving the "anterior closed space" of the terminal phalanx. With increasing tension in this area, the circulation is impaired and necrosis of soft tissues and bone occurs early. *B*, a paronychia (advanced: "run-around") an infection involving the nail fold and nail base

with vascular compression, resulting in a "felon" (Fig. 24, *A*). There is marked pain, throbbing and exquisite tenderness. Untreated, this process may lead to ischemic necrosis of soft tissue and osteomyelitis of the terminal phalanx. Decompression of the anterior closed space and drainage of exudate by incision may be required. It can be accomplished by use of a hockey-stick incision.

Paronychia.—A paronychia (Fig. 24, *B*) is a common infection of the nail fold, which may extend beneath the nail to form a *subungual abscess.* In the early stages of the infection a paronychia may

be treated conservatively. If the infection advances, it may be necessary to incise and reflect the nail fold and then to excise a portion of the root of the nail. The matrix should not be injured, because this may result in permanent deformity of the nail.

Furuncle, Carbuncle, Subepithelial Abscess.—These infections are treated as described under Furuncle, on page 147. A collar-button abscess is formed when two abscess cavities are connected by a narrow sinus They are often associated with calluses and commonly occur in the web of the hand.

MAJOR HAND INFECTIONS.—This group includes tenosynovitis, fascial space infection, acute cellulitis, acute lymphangitis and human

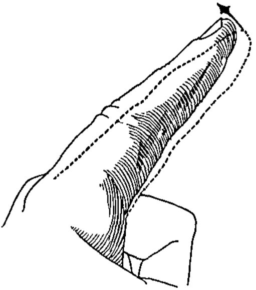

FIG. 25.—Tenosynovitis (tendon sheath infection) of the index finger and the cardinal signs of Kanavel (See text.)

bite infections. Special study is warranted because of the actual or potential seriousness of these infections.

Tenosynovitis.—A tendon sheath infection (Fig. 25), is a surgical emergency Rapid spread throughout the sheath may result in irreparable damage to the function of the hand. Tendon sheath infections commonly follow puncture wounds of the hand or finger. In addition to the usual signs of inflammation, there are four cardinal signs (Kanavel) of acute tenosynovitis: (1) fusiform swelling of the finger, (2) exquisite tenderness over the course of the tendon sheath, (3) fixed, flexed position of the finger and (4) severe pain on extension of the finger.

The flexor tendon sheaths of the hand follow a basic anatomic pattern (Fig. 26):

The tendon sheaths of the index, middle and ring fingers extend from the base of the distal phalanges to the level of the metacarpophalangeal joint or a thumb's breadth proximal to the webs. The sheaths of the thumb and the little finger extend from the base of the distal phalanges, pass beneath the annular ligament at the wrist and into the forearm, where they form the radial and ulnar bursae, respectively. The radial and ulnar bursae usually communicate.

Infections of the flexor tendon sheaths may extend to involve other spaces of the hand. The most likely extensions are:

1. From the sheath of the thumb to the radial and ulnar bursae.

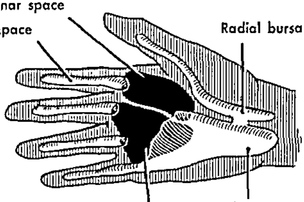

FIG 26 —Tendon sheaths, bursae and fascial spaces of palmar aspect of fingers, hand and wrist

The pus may point in the forearm (retroflexor [Parona's] space).

2. From the sheath of the little finger to the ulnar bursa, the radial bursa or the forearm
3. From the sheath of the index finger to the thenar space.
4. From the sheath of the middle finger and the sheath of the ring finger to the midpalmar space.

Unrelieved tension within the sheath causes vascular impairment and necrosis of the tendon The tendon sheaths can be drained by an incision placed at the side of the finger, but avoiding the digital nerves and blood vessels. The radial and the ulnar bursae and the retroflexor space can be drained by lateral incisions at the level of the wrist. Always consult authoritative works before making *any* incisions.

Fascial Space Infection.—The two major fascial spaces of the hand

lie in the palm between the flexor tendons and the interosseal muscles which cover the metacarpal bones. The *midpalmar space* lies in the ulnar half of the hand and the *thenar space* in the radial half. The spaces are separated by a layer of fascia attached to the third metacarpal bone. The fascial spaces may be infected directly from penetrating wounds, or indirectly by spreading cellulitis of the hand or from rupture of an infected tendon sheath.

Thenar Space Infection.—This infection (Fig. 27, *A*) is character-

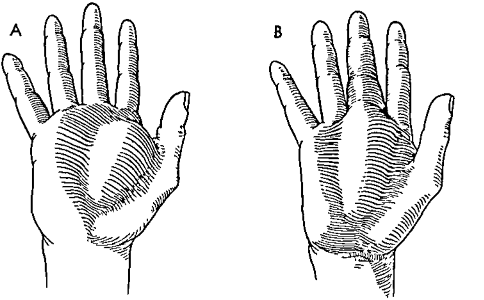

Fig 27 —Appearance of hand in deep space infections. *A*, thenar space abscess, *B*, midpalmar space abscess

ized by swelling of the thenar eminence, abduction with limitation of motion of the thumb and a bulging of the web between the thumb and index finger. The appearance is similar to that produced by clenching a golf ball in the radial side of the palm. The landmark for surgical drainage is on the dorsum of the hand parallel to the distal border of the first dorsal interosseal muscle.

Midpalmar Space Infection—Midpalmar space infection (Fig. 27, *B*) produces a fulness of the palm and loss of the normal concavity of the hand. Induration, swelling and tenderness may extend along the lumbrical canals into the webs of the fingers. A transverse incision parallel to the distal crease of the palm gives satisfactory drainage.

Retroflexor (Parona's) Space Infection.—The retroflexor fascial space lies in the forearm, between the flexor tendons and the pronator

quadratus muscle. It is rarely the site of a primary infection but may be involved by extension from the radial bursa or the ulnar bursa.

Cellulitis and Lymphangitis.—These conditions have been considered previously (pp. 148-149). Conservative measures are indicated. Surgical drainage is usually unnecessary and may be harmful.

The pattern of lymphatic drainage from the hand is:

1. From the anterior surface of the hand to the posterior surface of the hand
2. From the radial side of the hand directly to the axillary lymph nodes
3. From the ulnar side of the hand to the axillary nodes by way of the epitrochlear nodes
4. From the middle finger to the axillary lymph nodes, or to the nodes along the cephalic vein

Human Bite Infections.—Human bites are usually serious injuries. When virulent bacteria of the mouth are introduced into soft tissues or into joint spaces of the hand, satisfactory conditions for bacterial growth may lead to invasive infection. The inoculation often occurs when the clenched fist strikes a tooth (knuckle-tooth injury). The infection is polymicrobic and includes streptococci, staphylococci, Vincent's organisms and various anaerobes. If treatment is delayed, movement often disseminates the infectious material, and soon extensive inflammation and tissue destruction occur. Treatment consists of meticulous wound cleansing, wound excision, no closure, immobilization, warm packs (or activated zinc peroxide suspension), elevation, rest of the part and antibiotic therapy.

Osteomyelitis.—The bones of the hand are sometimes involved by extension from soft-tissue infections. The x-ray appearance may be misleading because the bone changes often appear more advanced than they actually are. The phalanges usually will regenerate after the infection has been overcome. For this reason, it is recommended that dead bone (sequestra) be removed and the soft tissues drained. Amputation for osteomyelitis of the fingers or hand is rarely necessary.

Special Infections of Surgical Importance

ERYSIPELAS.—Erysipelas is an acute spreading cellulitis and lymphangitis caused by hemolytic steptococci. The organisms gain entry through a break in the skin. A severe local and systemic reaction follows. The onset is often abrupt, with chills, fever, prostration and

lie in the palm between the flexor tendons and the interosseal muscles which cover the metacarpal bones. The *midpalmar space* lies in the ulnar half of the hand and the *thenar space* in the radial half. The spaces are separated by a layer of fascia attached to the third metacarpal bone. The fascial spaces may be infected directly from penetrating wounds, or indirectly by spreading cellulitis of the hand or from rupture of an infected tendon sheath.

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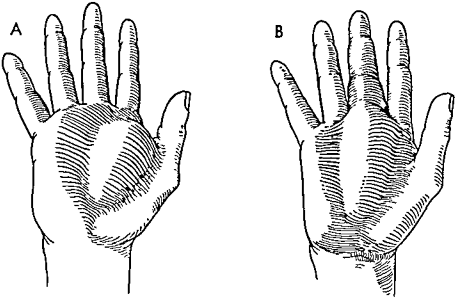

FIG 27 —Appearance of hand in deep space infections *A*, thenar space abscess, *B*, midpalmar space abscess.

ized by swelling of the thenar eminence, abduction with limitation of motion of the thumb and a bulging of the web between the thumb and index finger. The appearance is similar to that produced by clenching a golf ball in the radial side of the palm. The landmark for surgical drainage is on the dorsum of the hand parallel to the distal border of the first dorsal interosseal muscle.

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Retroflexor (Parona's) Space Infection.—The retroflexor fascial space lies in the forearm, between the flexor tendons and the pronator

cultured from the exudate. The lesions appear as multiple small pustules which extend and coalesce to form large areas of cutaneous gangrene and ulceration. Management is similar to that of postoperative gangrene, but favorable response is proportional to success in overcoming the primary disease.

FUSOSPIROCHETAL GANGRENE.—This is caused by mouth organisms such as fusiform bacilli, spirochetes, spirilla and nonhemolytic strepto-

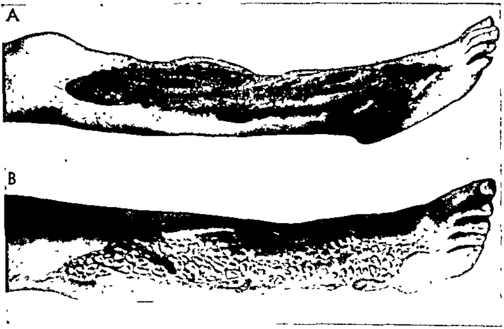

FIG. 28.—Synergistic gangrene of the leg due to mixed infection of microaerophilic nonhemolytic streptococcus and staphylococcus. *A*, after excision of necrotic tissue and before application of activated zinc peroxide suspension. *B*, healing after application of small deep grafts ("pinch grafts").

cocci often present in large numbers in dental caries. The infection may develop after a human bite injury (especially of the hand) or as a complication of lung abscess. Serious infections may be prevented by careful attention to the details of wound treatment. Fusospirochetal infection may occur in any wound which has been contaminated with mouth secretions, but it rarely develops unless there is associated tissue damage, extravasation of blood and delayed wound treatment with primary closure. When these conditions exist, the wound should be cleansed, excised (if possible) but not sutured (instead, left open). Established infections are treated by drainage, excision of devitalized tissue and local and systemic administration of antibacterial drugs.

rapid extension of the infection. The skin becomes fiery red, swollen and tender, and a distinct line of demarcation is observed at the advancing margins. Erysipelas commonly involves the face in a "butterfly" distribution over the cheeks and nose, but it also develops in other cutaneous areas. Blebs, lymphangitis and lymphadenitis appear, but suppuration is rare. In an extremity, repeated bouts may lead to chronic lymphedema.

The application of cool, moist compresses, coupled with intensive antibacterial therapy, is usually adequate treatment.

Acute Streptococcal Gangrene.—An uncommon infection also caused by hemolytic streptococci, but differing from erysipelas in several respects, is acute streptococcal gangrene. The onset of the infection is usually devoid of an alarming systemic reaction. The skin first becomes red, swollen and painful, then dusky and covered with blebs, and finally gangrenous.

In addition to the measures directed toward control of the infection, there may be need for early release of skin tension and ischemia by surgical incisions. Should gangrene occur, excision and subsequent skin grafting is often necessary.

Synergistic Infections
(Chronic Gangrene Group of Meleney)

Postoperative Progressive Bacterial Synergistic Gangrene.—This condition is caused by micro-aerophilic nonhemolytic streptococci in combination with staphylococci (Fig. 28). It may develop after abdominal operations in which wound contamination has occurred (e.g., ruptured appendicitis). Its onset may be delayed for several days, and serious systemic signs of infection are not prominent. Ultimately, an obstinate and progressive wound infection complicates healing and leads to gangrene of the tissues surrounding the wound. The peripheral zone of involvement is fiery red, the middle zone is purple, and the central zone is necrotic or black.

Vigorous treatment is indicated. The wound should be opened widely, cleansed by irrigation and débrided. A suspension of activated zinc peroxide or bacitracin may be applied topically. Systemic therapy is also necessary.

Gangrenous Impetigo.—*Gangrenous impetigo is most often found* as a complication in severe chronic debilitating diseases (e.g., chronic ulcerative colitis). Hemolytic streptococci and staphylococci can be

common, and the cure or arrest rate has increased, since the introduction of the new chemotherapeutic regimens (isoniazid, para-aminosalicylic acid [PAS] and streptomycin). Important advances in surgical treatment have also been developed, including segmental resection of the lung. The treatment of chest diseases is a special field of surgery and is not discussed in detail in this volume.

Tuberculous lymphadenitis is transmitted through raw milk from infected cows. The tubercle bacillus enters through the faucial tonsils or the lymphoid tissue of the lower ileum. The disease formerly was common in children and young adults, but its incidence has fallen with the widespread pasteurization of milk.

There may be few systemic signs beyond slight fever, weight loss and anemia. The cervical lymph nodes become enlarged, matted and fixed. Later they undergo caseation necrosis, ulcerate through the skin and form single or multiple discharging sinuses. Other aspects of tuberculous cervical lymphadenitis are considered under Masses in the Neck, in Chapter 25.

Intestinal tuberculosis may result from either the bovine or the human strain of Myco. tuberculosis. When intestinal infection is associated with pulmonary tuberculosis, it is usually caused by the human strain. In the ileum, the disease produces encircling or "girdle" ulcers, which may cause intestinal obstruction and other changes resembling regional enteritis. In the cecum, a hypertrophic form of the granulomatous process may simulate carcinoma. In the colon, the clinical picture often resembles ulcerative or amebic colitis or cancer.

Treatment must be directed at the primary site as well as the local process. Surgical excision of the diseased segment of bowel is often necessary.

Bone tuberculosis is generally blood-borne. Single or multiple foci develop, especially in spongy bone. The disease usually involves the vertebral bodies, the carpal and tarsal bones and the long bones. Typically, the process begins in the metaphysis, then extends to the epiphysis and joint. Diaphysial involvement is almost unknown.

Bone tuberculosis procedes at a relatively slow pace as destruction outdistances repair. The tubercles coalesce to produce large areas of tissue breakdown ("cold abscesses"), which may burrow beyond the primary site.

Tuberculosis of the spine (tuberculous spondylitis) is most common in childhood, and often develops between the midthoracic and midlumbar regions. Early diagnosis can be difficult before x-ray

AMEBIC INFECTION WITH GANGRENE.—Sometimes after drainage of amebic abscesses (usually hepatic) or following appendectomy in the presence of active amebic dysentery, gangrene sets in. It is caused by Endameba histolytica in association with other pathogens. The wound margins become elevated, everted and necrotic. The diagnosis is established by demonstrating the causative organism in suspensions of the exudate. The amebacidal drugs are effective.

UNUSUAL DISEASES OF ANIMAL TRANSMISSION

These infections should be reviewed by the student. Although rare, they are important clinically and should be kept in mind. They include: *rabies*, *erysipeloid* (usually of the hand in fishermen, butchers and veterinarians), *benign inoculation reticulosis* (cat-scratch fever), *tularemia*, *glanders*, and *anthrax*.

CHRONIC GRANULOMATOUS INFECTIONS

TUBERCULOSIS.—This disease more or less epitomizes the appearance and behavior of the granulomatous infections.

Human tuberculosis may be produced by either the human or the bovine strain of Mycobacterium tuberculosis. The human strain usually is responsible for pulmonary, intestinal, urinary, genital and peritoneal tuberculosis; the bovine strain, for bone and lymphatic tuberculosis. The organisms may enter the respiratory, alimentary or urinary passages and may spread by contiguity or through blood or lymphatic flow.

The general subject of tuberculosis, including the concept of immunity, the basic pathology, clinical manifestations, diagnostic technics and methods of treatment, should be reviewed elsewhere.

Final diagnosis will depend on demonstration of acid-fast organisms. Careful and repeated examination of exudates or tissues may be necessary before diagnosis is established. Guinea-pig inoculation is a most reliable test but has the disadvantage that it requires about six weeks. Special culture technics are helpful.

Tuberculous involvement of the lungs or bones may be established by x-ray examination. The skin tests (von Pirquet and Mantoux) may indicate sensitivity and give a clue to previous or existing infection.

Pulmonary tuberculosis and its complications have become less

are encountered: the common aerobic type, due to Actinomyces bovis, and the anaerobic type, due to Nocardia asteroides. Increasing attention is being paid to the latter as a cause of obscure chronic intraperitoneal and intrathoracic infections.

The usual portal of entry is the gastrointestinal tract. The infection is characterized by an unusual degree of granulation-tissue prolifera-

FIG. 29.—Actinomycosis of the jaw ("lumpy jaw"). Note the draining sinuses. The infection was cleared by intensive antibiotic therapy.

tion associated with secondary pyogenic infection, and suppuration. Frequently there is extension to adjacent fascia and bone but not into the lymphatics. Lymphangitis and lymphadenitis are not outstanding features of actinomycosis. Fistulas form, and a profuse exudate which contains small yellow bodies ("sulfur granules") is discharged. The diagnosis can be established by demonstrating the fungus in smears of the pus, in biopsy specimens or in cultures.

The infection is chronic in type and generally is not accompanied by a significant systemic reaction. Three clinical types are recognized: cervical, abdominal and thoracopulmonary.

changes occur. When the disease is suspected, serial radiographs should be made at frequent intervals, meanwhile treating the patient on the basis of the clinical diagnosis. Unless treatment is instituted, vertebral collapse occurs and an angular gibbus forms.

SYPHILIS.—Syphilis is caused by Treponema pallidum and has many clinical forms. Its late complications are most likely to come to the surgeon's attention. Since the advent of effective antibiotic therapy, serious complications of syphilis have become increasingly rare, but their importance in relation to the differential diagnosis of many conditions must be stressed.

In late syphilis, there typically is extensive tissue destruction, scarring and deformity. Focal necrosis (gumma) is the characteristic lesion. Any organ or tissue may be involved. Often the spirochete cannot be demonstrated in excised tissues. Serological tests are valuable aids to diagnosis.

Ulceration of the skin may occur. Leg ulcers are probably most common and usually appear over the anterior aspect of the tibial area and as single or multiple, shallow, ragged and "dirty" excavations. The ulcers must be differentiated from varicose, arteriosclerotic, saprophytic and neoplastic ulcerations. They do not respond to ordinary methods of treatment.

Gastrointestinal syphilis is uncommon, but occasionally luetic gastritis simulates diffuse scirrhous gastric cancer ("leather bottle," linitis plastica).

Vascular syphilis. The basic lesion in syphilis is an arteritis which appears in innumerable clinical forms. Of particular interest to the surgeon are aneurysms of the aorta and great vessels, some of which are now treated by operative repair, i e., resection of the aneurysm and segment of the vessel with reconstruction by use of an arterial homograft.

Skeletal syphilis appears chiefly as an osteoperiostitis. The infection is accompanied by a variable degree of bone destruction and repair. Differential diagnosis may be difficult and should include other specific types of infection, certain metabolic disorders and neoplasms.

Central nervous system syphilis also appears in many forms. One form is neuropathic joint disease (Charcot's), which can develop after trauma to or near a joint in a tabetic patient.

YEAST AND FUNGOUS INFECTIONS.—*Actinomycosis,* or "lumpy jaw" (Fig. 29), is the most important infection of this group Two forms

ASEPSIS, ANTISEPSIS AND DISINFECTION

Asepsis is concerned with the prevention of contamination of the wound by bacteria. It is accomplished by methods designed to remove or destroy bacteria on all objects which may come into contact with the wound. The aseptic technic involves an elaborate ritual of sterilization of all materials used in the operation: scrubbing of the hands and arms, covering the operator with a sterile gown and sterile gloves, masking the nose and mouth, preparing the skin of the operative area with antiseptics and other agents and decreasing the bacterial population of the operating room air as much as possible. There are many possibilities for "breaks" in technic, and the student must learn the applications and implications of this time-honored and important discipline.

Antisepsis refers to the use, on body tissues, of substances (antiseptics) which may kill or prevent multiplication of bacteria. In a broad sense the antibiotics may be considered antiseptics, although generally the term is reserved for chemical solutions.

Disinfection, or sterilization, implies the use of physical or chemical agencies to destroy bacteria which contaminate inanimate objects, such as instruments, surgical gloves, clothing, air, etc.

Sterilization can be accomplished by the use of moist heat under pressure, dry heat, ultraviolet and ionizing radiation, or chemicals such as the mercurials, phenols, cationic detergents, etc.

Agents Used as Surface Antiseptics

Although a great variety of agents have been used as "surface antiseptics" and the list is still increasing, no ideal antiseptic exists at the present time. All the chemicals that have potent and broad antibacterial action also have deleterious effects on tissues. Preparations which have recently become available have, however, simplified and made removal of bacteria from the skin and other body surfaces and from wounds more certain.

Formerly, soap and water, alcohol, ether, and tincture of iodine (or a mercurial antiseptic) constituted the common agents for preoperative skin preparation. Today, preparation of the skin is most commonly accomplished by prolonged washing with water and an antibacterial detergent soap containing hexachlorophene (or G-11), followed by the application of a cationic antiseptic in aqueous or

Treatment is both local and general. Long-continued administration of penicillin and sulfadiazine is most effective. Local drainage or excision may be necessary.

Blastomycosis (Fig. 30), which is less common than actinomycosis, is caused by the Blastomyces dermatitidis. There are two clinical forms: the cutaneous and systemic. The lesions appear as chronic, small papules which may ulcerate and are sometimes mistaken for furuncles. With systemic involvement, the lesions are widespread

Fig 30 —Chronic granulomatous lesions of the leg due to blastomycosis

and may resemble tuberculosis. Prognosis is poor. Diagnosis is established by demonstrating the organisms in exudate or biopsy material. Systemic administration of iodides and stilbamidine is recommended.

Less common infections in this group include: *coccidiosis* (coccidiodal granuloma), *sporotrichosis, histoplasmosis* and *torulosis.*

Lymphopathia Venereum.—This is a disease of virus origin which affects the lymphatic system primarily. It usually is localized in the pelvic region, and it occurs in both sexes. In women, there may be extensive pelvic lymphatic involvement, leading to esthiomene, a condition characterized by elephantiasis and ulceration of the vulva and anorectal structures. Late strictures of the rectum are common and must be differentiated from cancer The Frei test is an important diagnostic aid. Antibiotic therapy appears to be quite effective in arresting the disease.

Acetic acid solution (1–2 per cent) is commonly used for the treatment of pyocyanic (Pseudomonas aeruginosa) infections of wounds or burns. The dressing must be kept moist with the solution and changed daily, or more often if indicated.

Zinc peroxide suspension is prepared from medicinal zinc peroxide, a white powder. The powder, when activated by heating, liberates oxygen slowly if suspended in water or certain ointments. It is useful in the treatment of infections due to anaerobic (especially clostridial) and micro-aerophilic organisms. It has also been recommended for chronic undermining-burrowing ulcers, fusospirochetal infections, foul-smelling ulcerating carcinomas and perirectal abscesses.

Zinc peroxide is activated by heating to 140° C. for four hours. While dry, it remains active for about thirty days. Immediately before using, it is mixed with sterile water to form a creamy suspension. Sterile gauze saturated with the creamy suspension is placed in the wound, and the area is sealed off with sterile gauze strips impregnated with petrolatum or zinc oxide to prevent drying, caking and loss of oxygen. The dressing must be changed daily. Often a prompt change is noted in the character of the wound: the granulations become pink, the exudate decreases, offensive odors disappear and healing begins.

ANTIBIOTIC THERAPY

In surgery, as in medicine generally, many changes have followed the discovery and widespread use of antibacterial agents. The surgical infections, for the most part, can now be controlled. The management of the injured has become more predictable. Deformity, disability and death are less commonly the result of infection alone. Operations on contaminated or infected organs or tissues can be undertaken with increased safety. Skin-grafting procedures have been made almost uniformly successful. Peritonitis (e.g., in ruptured appendicitis) carries a much less serious prognosis. Intraperitoneal abscesses, pylephlebitis and septicemia are no longer common complications. Postoperative pneumonia, lung abscess and empyema can largely be prevented The new drugs also have made operations safer in areas formerly considered hazardous (e.g, bowel, lung). Finally, the antibiotics contribute a factor of time, which often makes it possible to defer operative treatment until systemic imbalances are corrected and conditions have become more nearly optimal for intervention.

The antibacterial agents have also brought new and different

tincture form (e.g., Zephiran,® Phemerol,® Cepryn®). The active principle in these solutions is a quarternary ammonium compound. These solutions are also used in the cleansing of wounds, but repeated applications must be limited because these agents produce disruption of tissue elements and hemolysis. The G-11 soaps (Phisohex,® Septisol,® etc.) have largely replaced "green soap" and the common white soaps in the surgical scrub.

Soaps aided by rubbing or scrubbing produce their effects through the emulsification and suspension of materials on the surface of the body. They have little antibacterial activity. Alcohol in a concentration of 70 per cent by weight is an efficient antibacterial agent and formerly was applied to the hand and operative area as part of the routine surgical preparation. At present it is considered unnecessary to use alcohol with the detergent soaps. It should be appreciated that ordinary soaps (anionic) effectively remove or neutralize the antibacterial hexachlorophene residue which remains on the skin after surgical scrubbing. Therefore, the cationic soaps should be used exclusively if the continued antibacterial action of hexachlorophene is desired.

Other substances are also used locally in the treatment of surface wounds. These are variously indicated for their cleansing, débridement (chemical) and antiseptic effects.

Sterile isotonic salt solution (0.9 per cent) is most widely used for wound irrigations and wet dressings. It is nonirritating, does not hemolyze red cells, prevents drying and crusting and allows drainage of the exudate into the dressing. When warm, it increases the blood flow locally and has some analgesic effect. Obviously, salt solution possesses no antibacterial properties.

Boric acid solution, in a 2–4 per cent concentration, is nonirritating and slightly antibacterial. Although unlikely, boric acid poisoning may occur from absorption after prolonged usage.

Magnesium sulfate solution (a 10–20 per cent hypertonic solution) is used in the treatment of open or closed infections in the stage of cellulitis. It has some analgesic properties and is thought to have some value in producing wound exudation.

Dakin's solution (sodium hypochlorite, 0.5–1.0 per cent) is an irritating alkaline chlorine preparation which exerts a destructive and solvent action on bacteria, pus, necrotic tissue and organic debris. It may be added to the dressings, directly or through irrigation tubes. The skin must be protected against its irritant effects.

granulation tissue and local thrombosis prevent free passage of the agents from the circulation into the area of the infection. Even at this time, however, specific therapy should be used in conjunction with surgical measures to prevent spread and aid resolution. Surgical treatment is concerned with the removal of the infected and necrotic tissue, drainage of the abscess, removal of foreign bodies, collapse of rigid abscess walls or cavities and closure of defects.

Infections respond best to antibiotic therapy when (*a*) the organism (or organisms) is "sensitive" to the drug; (*b*) there is an effective blood and lymphatic transport of the agent to the infected area; (*c*) the antibiotic effects are not nullified by inhibitory factors derived from the tissue or bacteria; and (*d*) the circulation and resistance of the host are normal. Should any of these conditions be lacking, the chances for a successful response to treatment are decreased.

Timing is important in the surgical treatment of infections. Operations should be undertaken, if possible, after antibacterial treatment has been started and before bacterial resistance has developed. It should be emphasized, however, that one must not depend on drugs to sustain the patient while delaying necessary surgical treatment. For example, patients with peritonitis due to ruptured appendicitis require combined specific drug and operative treatment.

A correct clinical diagnosis is important in the treatment of any infection An accurate bacteriologic diagnosis should be made if possible. Smears and cultures of the exudate should be obtained directly from the wound or from drainage material or aspirate from an abscess. Gram-stained smears are helpful in establishing a presumptive diagnosis before more complete bacterial studies are available. In protracted, spreading or chronic surgical infections a complete bacterial analysis, as well as in vitro antibiotic sensitivity studies, should be secured. In some instances (especially polymicrobic infections), it is necessary to utilize two or more antibacterial agents or to combine systemic and local treatment. It is important to recognize that changes in bacterial sensitivity, superinfection and growth of resistant organisms may occur during the course of the infection. For these reasons, periodic bacteriologic studies must be made when the response to treatment is not satisfactory or complications develop.

Prophylactic Use of Antibiotics

There is a definite field of usefulness for antibiotics in the prevention of invasive wound infections and an increased safety in the

problems. For example, there is the urgent need for *early diagnosis* and *aggressive therapy*, since delay favors the development of bacterial resistance, mixed infections and local wound changes which impair the effectiveness of these agents. Likewise, when antibiotics are given before the diagnosis has been established, "masking" may occur. For instance, when patients are given antibiotics for "virus pneumonia" when they actually have cancer of the lung, the opportunity for cure by operation may be lost. Another problem is represented by the overgrowth of nonsensitive organisms or fungi after antibiotic administration. Finally, the antibiotics have resulted in development of resistant strains of organisms which initially were sensitive (e.g., Staph. aureus).

Considerations in the Use of Antibacterial Agents

The two principal types of clinical response to infections are: wound suppuration and invasive infection.

Wound suppuration is evidenced by tissue necrosis, a surface exudation and little or no systemic response. Bacteria of relatively low virulence grow on a wound pabulum composed of blood clots and lymph, leukocytes and necrotic debris. Removal of the materials which serve to sustain bacterial growth, full mobilization of the humoral and cellular mechanisms which constitute the physiologic response to infection, and restoration of normal covering structures as early as possible are required. Antibiotics are helpful but not essential.

Invasive infection is characterized by local inflammatory changes with tissue necrosis and invasion of the adjacent tissues. There is a spreading cellulitis and lymphangitis. As extension of the process occurs, there is an associated systemic reaction. Here there is a dual problem: local treatment of the wound, and control of the invasive aspects of the infection. Systemic antibacterial therapy provides an indispensable adjunct to the treatment of invasive infections.

Specific therapy is most likely to control invasive infections in their early stages while there is an acute cellulitis. The effectiveness of these drugs, given systemically, depends in large measure on the integrity of the circulation. It is necessary that the drugs come into contact with bacteria in sufficient concentration to produce their effects. Furthermore, during the initial period of rapid bacterial multiplication, organisms are more susceptible to the effects of the drugs.

When an infection has been established for some time and has entered a subacute or a chronic stage, changes such as tissue necrosis,

4. Acute thoracopulmonary suppuration
5. Furuncles and carbuncles

In a second group, final recovery depends on a combination of properly timed antibacterial and operative treatment. This group includes:

1. Acute appendicitis with local peritonitis
2. Chronic osteomyelitis
3. Infections of the hand and foot
4. Bronchiectasis, empyema, lung abscess
5. Clostridial myositis (gas gangrene) and anaerobic cellulitis
6. Chronic granulomatous infections

Acute appendicitis with perforation and peritonitis requires prompt surgical care. Recovery from chronic osteomyelitis requires removal of dead bone (sequestra), obliteration of bone cavities and a covering of skin. Antibacterial therapy must be combined with surgical drainage for infections of the tendon sheaths, bursae and fascial spaces of the hands and feet. There is nothing that will justify the expectant treatment of gas gangrene, devitalized muscle must be excised and tissue tension must be relieved. Excision of the primary focus of infection is often necessary for the cure of chronic suppurative pulmonary disease. Excision of the major source of the disease may be required in tuberculosis and actinomycosis before recovery occurs.

Some principles which serve as a guide to the use of antibiotic drugs in treatment of surgical infections are:

1. The infecting organism or organisms must be sensitive to the particular agent used. Bacteriologic studies are indicated.

2. The concentration of the agent used must be maintained above the level of sensitivity of the infecting organisms to obtain a maximal bacteriostatic and/or bactericidal effect.

3. It is usually advisable to give large doses initially in order to minimize the hazard of drug resistance.

4. Mixed infections are common. Combined therapy with two or more agents is often necessary.

5. Local and systemic factors governing the control of bacterial infections must be given proper attention.

6. Operative measures must be carefully selected, timed and applied.

early post-traumatic or postoperative period. These drugs are used especially where the probability or possibility of bacterial contamination exists. It is generally unnecessary to administer antibiotics prophylactically to good-risk patients subjected to elective clean surgical operations (herniorrhaphy, thyroidectomy, etc.). In such cases the use of these drugs may be construed as a vote of "no confidence in aseptic technic."

Prophylactic administration of the antibacterial drugs is indicated in the following conditions:

1. Traumatic wounds and burns
2. Operations in contaminated areas (e.g., gastrointestinal tract, oral and pharyngeal cavities)
3. *Operations on patients with established infections* (e.g., respiratory, cardiac, gastrointestinal, genitourinary)
4. Emergency operations on patients with infections not related to the primary condition (e.g., combined acute streptococcal sore throat and acute appendicitis)
5. Elective or emergency operations when the condition of the patient makes pulmonary, urinary or other complications a definite threat
6. In certain other operations where, because of the magnitude and/or length of the procedure, the likelihood of infection is great (e.g., brain, chest, open reduction of fractures)

Active Treatment

Some surgical infections may respond to antibiotic therapy alone. Before tissue breakdown occurs, an effort should be made to establish high local concentrations of the drugs by systemic administration. Incision is harmful and accomplishes nothing in acute diffuse, spreading infections except when required for the release of tissue tension and the restoration of blood flow. Operative measures should generally be applied only when the infection has become localized and the indications for intervention are clear.

Antibacterial agents are of primary importance in the treatment of:

1. Cellulitis, lymphangitis, bacteremia, septicemia
2. Acute hematogenous osteomyelitis
3. Acute suppurative arthritis

clude their administration systemically except when their special antibacterial effects are required and measures are taken to protect the patient from these reactions.

Clinical Uses of Antibacterial Agents

Sulfonamides.—Preparations of the sulfonamides currently used in clinical practice include: (1) sulfadiazine, (2) Gantrisin® (sulfisoxazole), (3) Sulfasuxidine® (succinylsulfathiazole) and (4) Sulfathalidine® (phthalylsulfathiazole).

Sulfadiazine combines the features of minimal toxicity and maximal effectiveness in a variety of surgical infections. If penicillin is not available, sulfadiazine can be used with good results in hemolytic streptococcus infections. It has a low degree of activity against Staphylococcus aureus It may be used in combination with penicillin and streptomycin in the treatment of mixed infections. When the dosage is increased, there is increased toxicity, as with all chemotherapeutic agents except penicillin. Dosage is usually on the basis of a rule-of-thumb, depending on the severity of the infection, but for the average adult, 3.0–4 0 Gm. initially, and a maintenance dose of 1.0 Gm. every four hours is adequate.

Gantrisin® (sulfisoxazole) is widely used in the treatment of urinary tract infections. It possesses an unusual degree of solubility in the urine, even when the urine is acid, and is effective against many urinary tract pathogens. There is little danger of its crystallization in the urine, and it has no inherent toxicity. It may be given orally in doses of 1.0 Gm. three times daily, or more often if necessary.

Sulfasuxidine® and Sulfathalidine® *are used primarily as intestinal* antiseptics and are often called the "intestinal sulfas." These substances are not absorbed, and they are active against E. coli but ineffective against the intestinal streptococci. Given preoperatively, they effect a marked reduction in the coliform content of the stool and an increase in the safety of large-bowel operations. Sulfasuxidine® is the drug of choice, especially in the presence of partial bowel obstruction such as is often encountered in cancer of the colon. It produces a diarrhea-like stool and lessens the likelihood of complete obstruction. Sulfasuxidine® may be administered in the dosage of about 0.25 Gm./kg. body weight/24 hours, divided into six doses. The effective dosage range of Sulfathalidine® is one half of this schedule. These drugs may be combined with neomycin or other antibiotics for more

Local Use of Chemotherapeutic Agents

High bacteriostatic and bactericidal levels of antibacterial agents can be established by direct application of the agents to open wounds, skin and mucous membranes, gastrointestinal tract, paranasal sinuses, pulmonary tree, central nervous system and serous membranes. The *high expectations that were held for the local use of chemotherapeutic* agents (especially the sulfonamides) in clean and infected wounds, however, were dampened by the results of careful studies on wound healing in World War II. If the uses and limitations of local therapy are appreciated, it would seem that there is much to recommend a local drug level that is higher than would result from simple equilibrium with the plasma. The drawbacks to topical application of antibiotics include: allergic potentialities, tissue irritation and toxicity, the presence in the wound of inhibitory substances to some antibiotics and the difficulties associated with maintaining the drug in adequate concentration.

The agents most frequently applied topically include: bacitracin, neomycin and polymyxin B. Less commonly used substances include. *Sulfamylon,® Furacin,® gramicidin and tyrothricin.*

The sulfonamides are inactivated in the presence of pus and tissue extracts which contain a competitive biologic antagonist (para-aminobenzoic acid, PABA). Wounds which are bathed in a purulent exudate, therefore, cannot be effectively treated with sulfonamides.

While penicillin is effective in the presence of tissue debris and pus, it may be washed into the dressing by the exudate, and adequate local concentrations are difficult to maintain. Infections in closed cavities (e.g., joint space, the pleural cavity) may be treated with locally administered penicillin. Penicillin generally is ineffective against the gram-negative organisms which are commonly found in mixed wound infections. Penicillinase, an active penicillin-destroyer enzyme, is produced by Escherichia coli and certain strains of streptococci. At present, pencillin is not widely used as a topical agent.

Streptomycin is not recommended for local use. While the killing rate of streptomycin increases as the concentration is raised, bacterial resistance to the drug develops rapidly.

The broad-spectrum antibiotics generally have little place in local therapy because they produce local tissue irritation.

Bacitracin, neomycin and polymyxin B are suitable for topical application. They have certain neural and renal toxic effects which pre-

within forty-eight hours, other antibiotics (e.g., broad-spectrum drugs, erythromycin) may be indicated.

3. In clostridial myositis (gas gangrene): 1,000,000 units of crystalline penicillin every three hours has been recommended.
4. In the prevention or treatment of peritonitis or mixed infections due to enteric pathogens: 300,000 units of procaine penicillin G combined with 0.5 Gm. of streptomycin twice daily.

STREPTOMYCIN.—The antibacterial spectrum of streptomycin is restricted, but it supplements that of penicillin to good advantage. There may be a synergistic effect wtih penicillin. Streptomycin is effective against many gram-negative bacilli, especially E. coli, Ps. aeruginosa and B. proteus, as well as Myco. tuberculosis. In the treatment of tuberculosis, streptomycin is combined with PAS and other agents with great effectiveness.

For most infections, streptomycin has been replaced by other drugs. The most important reasons for this trend are (1) other agents do all that streptomycin can do, and do it more effectively; (2) bacterial resistance to streptomycin develops rapidly and (3) there is a definite neurotoxicity associated with the prolonged use of streptomycin. Permanent damage to the vestibular portion of the acoustic nerve and permanent nerve deafness may occur. Dihydrostreptomycin is somewhat less toxic. In acute infections the usual dose is 0.5 Gm. intramuscularly every four to six hours.

BROAD-SPECTRUM ANTIBIOTICS.—The following are the broad-spectrum antibiotics in common use: (1) chlortetracycline (Aureomycin®), (2) chloramphenicol (Chloromycetin®), (3) oxytetracycline (Terramycin®), (4) tetracycline, (5) erythromycin and (6) carbomycin. These antibiotics have been grouped together because they have much in common, so far as their source and clinical usage is concerned. Each was originally derived from a species of Streptomyces. They all inhibit gram-positive, as well as gram-negative, organisms. They all have antiviral properties and are active against some bacteria which are resistant to penicillin and streptomycin. They have a relatively low toxicity for man and animals and they can be given by mouth. The side effects produced by them include: nausea, vomiting, diarrhea and proctitis, and occasionally hypersensitivity. Rarely, chloramphenicol produces bone-marrow depression with leukopenia or aplastic anemia and death. The danger of such reactions is slight, provided that the patient is followed carefully. When in vitro sensitivity tests indicate the need for chloramphenicol, it should be used.

complete and rapid reduction of the bowel bacteria. Prolonged administration of intestinal antiseptics leads to disappearance of vitamin K-producing coliform bacteria and a reduction of plasma prothrombin. For this reason, it is recommended that parenteral vitamin K be given before operation, to correct a possible bleeding tendency.

PENICILLIN.—At present, penicillin is the most useful and most widely used antibiotic agent. It has a high level of potency, is almost nontoxic and is cheap. It is the antibiotic of choice in the treatment of all gram-positive infections, including those of staphylococcic, pneumococcic and streptococcic origin. It is also effective in the treatment of gonorrhea and syphilis. It has the disadvantages of poor gastrointestinal absorption, rapid renal excretion and poor penetration of the blood-brain barrier. As mentioned before, it is inactivated by penicillinase. Allergic and toxic manifestations to penicillin administration are infrequent (about 5 per cent) but are potentially serious.

The penicillin preparations commonly used include: (1) procaine penicillin G (for aqueous injection), (2) crystalline potassium penicillin G (for aqueous injection), (3) potassium penicillin tablets (for oral administration) and (4) procaine penicillin G in streptomycin or *dihydrostreptomycin sulfate solution.*

There are certain general principles in the use of penicillin which should be kept in mind. (1) The organism causing the infection should be known. If response to treatment is not prompt, the sensitivity of the organism should be tested. (2) The tissue concentration of penicillin should be adequate to inhibit completely the growth of organisms. (3) It is desirable to maintain a minimum effective concentration of penicillin in the tissues at all times until the patient has recovered.

There are no established dosage levels for penicillin therapy. The general tendency is to give unnecessarily large amounts because of its low toxicity. Meleney has stated that, if penicillin is going to be effective, it will probably be effective with doses not exceeding 200,000 units a day. In general, the following dosages are used:

1. For prophylaxis in the treatment of wounds and burns, or in the prevention of postoperative complications: 100,000 units of crystalline penicillin G combined with 300,000 units of procaine penicillin G daily, or twice daily.
2. For serious surgical infections: 500,000 units of crystalline penicillin G every eight hours, or the combined aqueous and procaine preparation every twelve hours. If there is no response

for intramuscular injection. This drug likewise has some degree of renal and nerve toxicity when given parenterally. It should never be given intravenously.

Neomycin is widely used for topical application to infected surfaces. It may be applied as a wet dressing in a concentration of 5.0 mg./ml., or in powder or ointment form. It is also effective against many gram-negative organisms. It must not be given parenterally. Oral preparations are effective against the intestinal bacteria, and neomycin is often used in combination with sulfonamides preoperatively.

It should be pointed out that dilute organic acids (citric, lactic and acetic), certain detergents, and hexachlorophene soaps also have antibacterial properties and are useful in suppressing saprophytic infections. It may be unnecessary to resort to the topical antibiotics when wound contamination or surface infection exists. Much can be accomplished by simple clean care and the application of less expensive and probably equally effective agents.

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The broad-spectrum antibiotics are available in capsules containing 50–250 mg each. The dosage varies from 1.0 to 2.0 Gm daily, given in divided doses every four to eight hours. Except in severe infections, 750 mg to 1 Gm. daily is adequate. When intravenous administration is required, the special preparations and diluents provided by the manufacturer must be used and the instructions followed carefully. Both chlortetracycline and oxytetracycline have been recommended for bowel preparation, and both are effective in the treatment of peritonitis.

Tetracycline has been available for clinical usage since 1953. Its antibacterial spectrum is similar to that of chlortetracycline and oxytetracycline, and it is said to be better tolerated. It is probable that tetracycline will replace the older agents in time. The dosage and routes of administration are the same as for the related drugs.

Erythromycin and carbomycin also became available in 1953 Both are effective against the gram-positive bacteria, certain large viruses and the rickettsias. Erythromycin is most useful when the infection is due to penicillin-resistant gram-positive cocci or when the patient is sensitive to penicillin The place of carbomycin in antibacterial therapy has not as yet been established.

ANTIBIOTICS USED TOPICALLY IN SURGICAL INFECTIONS.—Bacitracin, polymyxin B and neomycin are commonly used for topical application.

Bacitracin has a broad bacterial spectrum and is bacteriocidal in direct proportion to its concentration. It is effective against staphylococcal infections which have become resistant to penicillin. It is not inactivated by penicillinase. It has no locally irritant effect and little tendency to produce allergic reactions. Systemic administration of bacitracin is limited by nephrotoxicity, but serious renal damage can be avoided or prevented by proper regulation of dosage. Bacitracin is used topically in concentrations of 100–500 units/ml. If antibiotic sensitivity studies indicate the need for systemic bacitracin, intramuscular injection of 20,000 units three to five times daily, combined with penicillin, is recommended

Bacitracin is only slightly absorbed from the gastrointestinal tract. It has been recommended for preparation of the bowel prior to resection of the colon. It may be combined with neomycin or the intestinal sulfonamides

Polymyxin B (Aerosporin®) is effective against a wide variety of gram-negative organisms, including Ps. aeruginosa. The drug is used topically in a concentration of 1.0–2.5 mg./ml. It is also available

CHAPTER 8

Thermal Burns

AS WITH any injury, a complex sequence of general and local reactions is initiated at the moment of burning. An old biologic law is reaffirmed: nothing changes by itself, in vacuo, apart from the whole. So, the burn problem is essentially twofold: the patient and the burn wound; general care and local treatment. Moreover, the nature of the local changes in the burned area and the systemic reactions to burning are not static. Changing pathophysiologic mechanisms operate in a framework of time. The orientation of treatment in time-space is as fundamental as basing treatment, in the first place, on an understanding of the anatomy of burning and of pathophysiologic mechanisms. For example, "too much—too early" may be, in certain situations, as fatal to the patient as "too little—too late."

Infinitely variable in detail, the local care of the burned area must not be allowed to deviate from basic surgical principles of wound treatment. The burn is an open wound, not unlike other traumatic wounds, and the aim of therapy is to convert it without delay into a clean and healing wound. Nothing can be done which will accelerate healing; yet everything must be done to avoid delaying it. Under favorable conditions, the burn wound will heal primarily, without sepsis, if partial-thickness skin loss has occurred or secondarily if full-thickness loss has taken place.

The general reactions to an extensive body burn are complex and incompletely understood The approach to the burned patient will be chaotic and confused unless it is based on modern concepts of cardiovascular dynamics and shock, water and electrolyte balance, total nutrition, respiratory physiology, the metabolic responses to stress and burning, endocrinology and surgical bacteriology. Such is the

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permeability and loss of fluid from the surface and into and around the burned area.

In a *third degree burn* there is complete destruction of the skin and its appendages, as well as of deeper structures. Healing occurs only after the dead burned tissue has been removed and epithelium has been regenerated from the wound margins or the area is covered by skin grafts. A third degree burn may be less painful than a second degree burn because nerve endings are destroyed rather than exposed. In third degree burns the surrounding areas usually sustain some second and first degree damage. In most burns the burned area varies greatly in size and depth. Infection is always a problem in serious burns. Dead tissue is an ideal pabulum for the growth of the resident skin bacteria and the transient organisms which may be introduced.

Initially, it may be impossible to determine with accuracy the relative extent of second and third degree injury unless the exact agency and exposure is known. Therefore, in the presence of an extensive burn, one must assume that the injury is serious and that treatment for prevention of shock is required. It is a common error to underestimate the depth of the burn. The clinical measurement of the surface area involved is based on the "rule of nines" (Fig. 31). One may calculate each upper extremity to represent 9 per cent; each lower extremity, 18 per cent, the anterior and posterior surfaces of the trunk, each 18 per cent; and the head and neck, 9 per cent. In infants and children these estimations must be corrected according to age, giving a higher percentage for the head and smaller percentage for the extremities.

Local Effects

After severe burning, there are various gradients of injury at different levels in the burn wound. The damage which results may be either reversible or irreversible.

The special significance of *irreversible* tissue changes in the zone of injury relates to the immediate survival problem of burn shock and the systemic requirements of the burned patient. The extent of the burn, in terms of surface area, is related directly to the immediate mortality from burn shock in the first forty-eight hours or so.

The severity and type of injury at different levels in an extensive burn wound can be described as follows:

1. At the surface, irreversible tissue injury and cell death by charring or coagulation-necrosis follow critical exposure to high tem-

broad framework supplementing the technical know-how of treatment. Even so, survival is unlikely should the surface area of the burn approximate 50 per cent.

Burns are generally considered according to the depth of tissue injury and classified into first, second and third degrees.

A *first degree burn* produces simple cutaneous erythema and edema. It involves only the must superficial layers of the skin. There

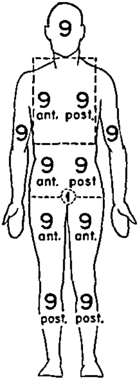

FIG 31.—Estimated percentage of surface area of the adult body according to the "rule of nines." In infants and children, the head represents a relatively larger percentage, and the extremities a smaller percentage, of the body surface.

is minimal penetration of the epidermis, and the problem of infection is generally negligible Such a burn may be quite painful, but it heals rapidly with or without treatment. Serious systemic effects are rare unless the burn is very extensive.

A *second degree burn* is characterized by blistering. It involves varying thicknesses of the skin, but either a portion of the dermis or epithelial islands of sweat gland and hair follicles remain viable. Because of this, unless further damage occurs from misguided therapy or infection, regeneration of the skin occurs rapidly. In a burn of this depth, there is damage to deep tissues, causing increased capillary

laries into the intercellular fluid, causing decrease in plasma volume. Although maximal in the first six to eight hours, a relentless loss of plasma-like fluid with high electrolyte content continues until an equilibrium is reached after thirty-six to seventy-two hours. The shock cycle of oligemia is initiated. All mechanical and pharmacologic means which have been attempted to decrease abnormal capillary and cell-membrane permeability in the zone of injury have proved valueless. The volume of fluid trapped in the so-called "third space" is roughly proportional to the area of the surface burn, since surface area gives at least an estimation of volume of tissue involved. Surface losses from the burned area, continued water loss by vaporization and renal requirements contribute to the water-electrolyte deficit which develops. Plasma loss occurs as long as there is leakage of fluid into the interstitial spaces. *Water* and *electrolyte* requirements must be met. *Red cell mass* is lowered, particularly in deep extensive burns, by a combination of factors: intravascular hemolysis from heat, depression of red cell formation, wound hemorrhage and surface infection, altered iron and globin metabolism.

A danger period occurs with the shift of fluid back into the vascular compartment at forty-eight to seventy-two hours. At this time, *resorption of edema fluid* is a basis for cardiovascular overloading. The maximal expansibility of the extracellular fluid has taken place, equilibrium has been established, and a *diuresis* should be watched for. Also, with vascular compartment overloading, as fluid shifts back from the third space into the circulation, a *dilutional anemia* develops. Transfusion at this time may kill by an overload.

Lung Burn and Pulmonary Sequelae

The inhalation of smoke, fire itself or gas may create a respiratory emergency. Rapid respirations, cyanosis, rhonchi and râles, and visible evidence of burning around the face and lips should suggest this possibility. The basic pathologic changes which occur are hemorrhagic or necrotizing laryngotracheobronchitis with pulmonary edema, upper-airway obstruction and, if the patient does not die at once, atelectasis, pneumonitis and pulmonary infarction.

Hyperpyrexia

Seen in the first twenty-four hours after burning, when there are signs of anoxia of the central nervous system, fever may be related

peratures—whether the temperatures are from radiant, wet or dry heat.

2. Deep to this zone of total destruction will be an area of intravascular thrombosis and varying degrees of hemolysis. Red cell hemolysis may lead to hemoglobinemia and hemoglobinuria.

3. More remote from the burned surface, the serious (but reversible) consequences of burning take place. Large volumes of tissues are involved, and surface-area estimates are the best available basis for clinical evaluation of the degree of leakage of plasma-like fluid, which takes place relentlessly for a period of some forty-eight hours through damaged capillary membranes. The pathologic shift of plasma volume with trapping of plasma-like fluid in the extracellular space of the burned area is largely responsible for burn shock. Alterations in cell membrane permeability also result in abnormal internal shifts of sodium and potassium between the tissue cells and the extracellular fluid. Wound edema progresses during the acute phase after burning, and as much as 10 L. or more of plasma-like fluid may be unavailable to the general body economy during this time. The replacement requirement is obviously large. Burn shock, if it develops, is characterized by reduced blood volume, hemoconcentration and high blood viscosity. Resorption of edema fluid occurs as the normal permeability characteristics of capillaries in the damaged area return.

The local evidence of these fluid alterations is blistering, if the injury is near the skin surface, and wound edema, if the deep tissues are involved. Except for a lowered protein content, blister fluid and lymph draining from the burned area resemble blood plasma. Since the damage which occurs is nonselective, not only is fluid lost into the extracellular space, but direct inoculation of the blood stream with bacteria and pyrogens can occur. The adverse local effects of wound edema are circulatory embarrassment and tissue anoxia. Edema fluid having high protein content is an irritant which, in itself, produces an inflammatory response. In special anatomic areas, such as the respiratory tract, unique risks from wound edema exist.

4. *At the limits of the zone of injury, a slight inflammatory response* with capillary dilatation and increased blood flow is the only evidence of injury.

Systemic Effects (Burn Shock)

Contributions are made to burn shock by pain, exposure and loss of body heat, but the significant factor in the production of shock is an internal shift of plasma-like fluid across heat-damaged capil-

3. *Starvation, sepsis* and *negative nitrogen balance* are the critical factors if the first two phases are survived, until the burned area is entirely healed. Within three to four days after burning, the *total nutritional requirements* of the burned patient for water, electrolytes, red cell mass, vitamins, the energy requirement (carbohydrate and fat) and protein must be under constant scrutiny. Stress, starvation, the catabolic response, and exudate losses all contribute to negative nitrogen balance. As the burn is covered, exudate nitrogen losses decrease. With oral intake, following the resorption of edema fluid, zero sodium and potassium balance are maintained with dietary intake. A large loss of body protein demands replacement of red cell mass: 500–1,000 ml. of whole blood each week may be necessary until the patient is clearly in positive nitrogen balance; with sepsis controlled, and skin grafting under way, recovery begins.

The problem of *nutritional sustenance* is a critical factor in survival.

4. *Positive nitrogen balance* and *convalescence* are almost the direct linear functions of nitrogen and caloric intake. As soon as burn shock has been corrected, the intake should be increased to high levels by tube feeding, if necessary. (See Chapter 5, on Nutritional Balance.) Nutrition becomes the key to recovery, and the so-called *stalled convalescence* (Moore) of stationary progress (continued weight loss, sepsis, failure of the grafts to take, and progressive decline) follows if the trend is not reversed. It is the major mortality factor of this period. Mortality in this phase is frequently due to physician-failure.

Gastrointestinal Effects of Burning

The oral administration of fluids during the shock period is often not feasible if there has been major burning, for nausea and some vomiting are the rule. A particular problem of the burned patient (which sometimes may have fatal results) from acute perforation or exsanguinating gastrointestinal hemorrhage is Curling's ulcer, the so-called "stress ulcer" of the stomach and small bowel and duodenum. It is apparently related to endocrine factors and mobilization of the pituitary-adrenal axis by the stress of burning.

Renal and Hepatic Injury

Renal and hepatic injury may result from anoxia due to burn shock, from absorption of toxic breakdown products into the circulation or

to a variety of factors: (1) the hypermetabolic response of stress and burning, (2) extensive skin loss with failure of heat regulatory mechanisms, (3) absorption of pyrogens into the circulation through the damaged capillary membrane in the burned area and (4) acute liver failure. Most often, however, it is an indication of *sepsis* and suggests the presence of full-thickness skin loss. If there is anorexia, rapidly developing anemia and dressings which are soaked with smelly exudate, cleansing, early débridement and skin grafting are indicated.

Biologic Response to Stress

The totality of the *metabolic changes* occurring in the burned patient has been summarized by Moore (1951) as consisting of (1) *immobilization,* (2) *starvation* and (3) *endocrine response* of pituitary-adrenal activation. The metabolic course of a severe 25–50 per cent surface area burn is similar to other severe injuries and will follow four phases, about as follows:

1. *Shock and sodium retention, stress and starvation,* mark the first phase of four to six days. There is relentless expansion of the ECF because of capillary and cell membrane injury 10–15 L. of high-electrolyte plasma-like fluid collects as wound edema. During this period, there is almost total starvation except for the small amounts of dextrose given intravenously. There is a relative, but reversible, renal insufficiency from adrenal effects on the nephron and from the changes in renal hemodynamics occurring with burn shock. The metabolic pool of protein is called upon to meet the energy requirement. There may be as much as 1,000 mEq. positive sodium balance (wound edema) and a 150 or 200 mEq. negative potassium balance, owing to starvation.

2. *Sodium diuresis* and *resorption of edema* are the first major readjustments, occurring forty-eight to seventy-two hours after burning, if initial burn shock has been survived. Evidences of activation of the pituitary-adrenal axis persist: alterations in renal excretion, eosinopenia, increased 17-ketosteroid excretion, etc., but at a critical point the balance shifts, the capillary membrane reconstitutes its normal permeability characteristics and a water-sodium diuresis of edema fluid begins, as edema fluid shifts back into the vascular compartment. This is the danger period of water overload. Increased renal excretion of water, in excess of 100/ml. hour, points out the need for caution in parenteral therapy.

in an effort to lower the hematocrit to more nearly normal levels.

Experience has shown that the most satisfactory guides to therapy are: the urinary volume output, the urine specific gravity and the clinical condition of the patient. Other things being equal, as long as there is an adequate urinary output, *there is no need to be greatly* alarmed; but if the output falls markedly or ceases, trouble can be expected. The seriously burned patient should have an indwelling catheter inserted into the bladder, and the urinary output should be measured and recorded every hour. A rate of 30–50 ml./hour is considered optimal.

Although some clinicians prefer plasma transfusions in the early treatment of burned patients, most favor whole blood in adequate volumes. Hemoconcentration as a result of red cell overtransfusion has not been shown to be the cause of death in burn patients.

It is safer to anticipate shock and begin treatment than to await unequivocal signs of shock. Homeostatic mechanisms may support the patient for a time, and then fail rapidly. As it is basic to all medical therapy, so it is with shock: it is easier and more desirable to prevent rather than to treat.

Equal volumes of whole blood and ECF-like solutions should be given. The electrolyte solution consists of 0.9 per cent sodium chloride and M/6 sodium lactate in a 4:1 ratio Plasma expander (dextran derivative) in the amount of 500 ml. is acceptable in the adult patient. If the burn is not extensive and the patient responds satisfactorily, this may be all the blood or blood substitute required. Following these substances, the saline-lactate mixture may again be given. The quantity of the saline-lactate mixture should rarely exceed 1,500–2,000 ml. *in twenty-four hours; otherwise the tendency to edema is likely* to be increased. Glucose, 5 per cent in water, is given to supply metabolic needs.

As soon as the patient is able to take fluids, the oral route should be used: a solution consisting of 1 teaspoonful (or 4 Gm.) of table salt and ½ teaspoonful of baking soda added to a quart of water will provide needed electrolytes and will obviate the danger of fluid overload. Because the quantity of salt is small, it will not cause nausea and can be taken instead of plain water. The mixture may be flavored with raspberry or a similar substance to make it palatable.

If the urinary output is satisfactory and the specific gravity is low, the rate of fluid administration may be decreased. If the urine specific gravity remains high, the rate of flow should be maintained or in-

from bacteremia and established sepsis. Hemoglobinuria also occurs. Almost all kidney shutdowns in the burned patient are physiologic oligurias from adrenal factors and circulatory effects of inadequate blood volume replacement, and are reversible with time and adequate shock therapy. Acute renal failure is uncommon. *Hepatic necrosis* is rarely seen. Tannic acid therapy, which was formerly popular, produced a relatively high incidence of liver damage, owing to tannic acid absorption from the burned surface.

Treatment of Burns

A small, simple burn is easily treated with petrolatum gauze and a dressing. It will heal rapidly provided that the healing process is not disturbed by repeated changes of dressings or the application of harmful substances.

Severe second or third degree burns, however, are different problems. The first objective is to keep the patient alive during the critical phase of the shock (forty-eight to seventy-two hours). An estimation of the percentage of burned body surface should be made. The depth of the burn will vary in different areas, and gross evaluation is usually adequate. It is usually better to overestimate the seriousness of the burn than to underestimate it.

If the patient is not in shock, blood is drawn for typing and cross-matching, and an intravenous infusion is started. If the patient is already in shock, it may be necessary to cut down on a sizable vein (usually at the ankle) and insert a cannula, preferably of the polyethylene type. If the patient is in impending shock or established shock, a plasma expander is started. It is important that blood be withdrawn for cross-matching purposes before the expander is administered. Otherwise there is likelihood of cross-matching difficulties later.

Until recently, the hemoglobin level, red blood cell count and hematocrit value were used as guides to the administration of blood and other fluids. It has been found, however, that these indexes in themselves are often of little value and may even lead to dangerous errors. While it is true that the fluid portion of the blood is lost in greater quantity than the red blood cells during the acute postburn period, there is also an inevitable loss of red cells, which in some instances may be extreme. The hematocrit may remain elevated for five or six days after the burn has been incurred. If the hematocrit is followed, there is a dangerous temptation to give excessive fluid

a small woman), and (2) the location of the burn (e.g., burns of the buttocks or scrotum may lead to sequestration of larger quantities of fluid than in burns of other areas). There are no formulas which can take the place of good clinical judgment.

Local Treatment of Serious Burns

When shock treatment has been instituted and when the patient has responded favorably, attention should be turned to the local treatment of the burns.

The *"exposure" and "open" treatment* of burns has recently been advocated by some surgeons. This method depends on the fact that after several hours the serum exuding from the burned surface coagulates and forms an eschar or covering crust, which seals off the area. The exposure method, however, has certain drawbacks. For example, if there is a circumferential burn of the trunk or extremity, the dependent surface usually becomes adherent to the bedsheets and the burn remains wet. The eschar may peel off when the patient moves. In some regions (e.g., extremity) this difficulty can be avoided if the part is suspended and kept dry. If properly used, the exposure method probably leads to less maceration and loss of viable tissue than the closed method.

After the initial period, pain also appears to be less intense when the burn is permitted to dry in the air. There is also some evidence indicating that the febrile reaction is less and that the patient feels better and has a better appetite when treated by the open method. However, later, when the dry eschar begins to crack, especially around the edges, dressings are necessary to prevent further bacterial contamination from the outside The exposure method appears to have its greatest usefulness in extensive first and second degree burns, which usually heal rapidly regardless of the type of treatment used.

The *"closed" method*, or covering of burn sites by bandaging, is usually preferable provided that it is used in a sensible manner. It is not generally practicable to bandage the face, genitalia or the perineum, because these areas rapidly become soaked with exudate and secretions, producing maceration. Burns in these regions are best handled by simply applying petrolatum gauze as often as necessary or by leaving them exposed.

The local treatment of a serious burn is shown in Figure 32. However, it is important that burn treatment be individualized and flexible

creased If the urinary output falls below 25 ml./hour in spite of a rapid infusion (120 drops/minute), blood should be given again, because this may be warning of impending shock.

In the event of mass catastrophe, it would be impracticable to follow the hourly urinary output and to give individual attention to each burn casualty. Under these circumstances, the formulas given in Table 12 may be used as a guide to blood and fluid needs.

TABLE 12.—SURFACE AREA ESTIMATE OF REQUIREMENT OF BLOOD-PLASMA EXPANDER AND ELECTROLYTE SOLUTION

DAY OF BURN

a) 1.0 ml of blood-plasma expander/kg. body weight/1% surface area burned, in the first 24 hours.
b) 1 0 ml. balanced electrolyte solution/kg. body weight/1% surface area burned in first 24 hours.
c) 1,000–2,000 ml. 5% glucose in water for metabolic needs.

NOTE· Do not give more than 8–10 L /24 hours, regardless of the extent of the burn.
First 8 hours ½ fluid volume, then ¼, ¼ in subsequent periods. Anticipated renal response: 30–50 ml./hour. Increase the rate of infusion if there is oliguria, decrease rate of infusion if more than 50 ml /hour.

SECOND DAY

Reduce the blood and electrolyte administration to ½ or ¾ of the burn-day estimate.
2,000 ml. 5% glucose in water for metabolic needs.
Volume limitation: about 5 L.

The calculated volume should consist of whole blood, blood plasma or plasma expander and an equal volume of electrolyte solution. In addition, 1,000–2,000 ml. of 5 per cent dextrose should be administered during the first twenty-four hours after burning. During the second twenty-four hours, one half to three fourths of the calculated quantities should be given.

The requirements for a 60 kg. patient with a 30 per cent burn are: $1 \times 30 \times 60 = 1{,}800$ ml. Thus, 1,800 ml. of blood or of blood and blood substitute, 1,800 ml. of electrolyte solution and 5 per cent dextrose in amounts from 1,000 to 2,000 ml. should be given in the first twenty-four hours. One half of this quantity of blood and electrolyte solution and 1,000–2,000 ml. of 5 per cent dextrose should be given in the second twenty-four hours While formulas of this type have a place in therapy, it should be recognized that all biologic phenomena are subject to so many variables that generalization is dangerous. Some obvious variables are: (1) the effect which burns of various depth have on the development of wound edema (thus, the same percentage of burned body surface in a large man is not comparable to that of

venous and lymphatic stasis but has a negligible effect on the development of edema. If pressure is applied for the purpose of preventing edema, there will be interference with blood flow and fluid will collect in areas beyond the pressure dressing. Improperly applied pressure dressings have sometimes endangered life and limb. For example, if a pressure dressing is applied to a burn of the forearm, leaving the hand and wrist free, marked edema and venous congestion of the hand soon become evident. In such an instance, contracture (resembling Volkmann's ischemic contracture) may develop if the dressing is allowed to remain in place over a period of days. If pressure dressings are used, the bandage should always incorporate the distal part of the extremity. Burns of the hand require that each finger be covered separately and the entire hand then wrapped in the "position of function." On the chest or abdomen, circular compression dressings should be avoided, or applied with great care, in order that they produce no disturbances in ventilation or circulation.

All burned patients must receive tetanus antitoxin (at least 3,000 units) or a booster dose of tetanus toxoid if active immunization has been previously established. Topical antibiotics are not generally used. Systemic administration of antibiotics (usually penicillin) should be begun immediately and continued as needed. The delayed coagulation of burn exudate favors development of high local concentrations of the antibiotics in the early postburn period.

Postburn Treatment

Burned patients who have been exposed to noxious gases or who have lost consciousness during a fire are prone to develop pulmonary complications. They are best treated in a highly humidified room, preferably of the "cold" type. Antibiotic therapy should be vigorous in order to aid in the prevention of these complications. Oxygen is often essential. Repeated aspiration of tracheobronchial mucus is indicated. If there is airway obstruction (manifested by stridor, retraction of the chest with difficult breathing, apprehension, cyanosis or restlessness), tracheotomy should be performed *without delay*. The respiratory difficulty is usually overcome in five to six days if care is vigilant and sustained.

When the patient has a marked rise in body temperature, every effort must be made to reduce the fever promptly (see Chapter 30, on Head Injuries). A sustained elevation in the range of 105° F.

and that those conditions which may lead to further tissue damage be avoided. Cleansing of the burned areas with either a detergent or a mild soap, rinsing with saline solution, opening of the blisters and removing loose dead tissue has been accepted practice. However, these measures may lead to increased local damage and do not pre-

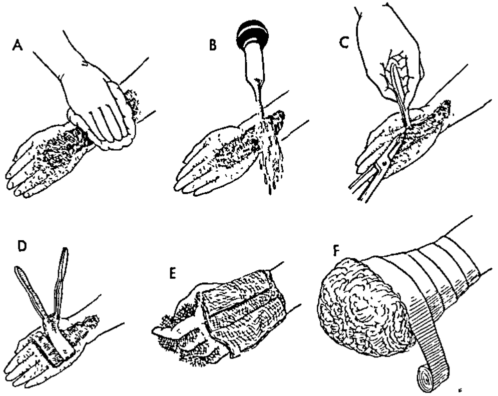

FIG 32.—Steps in the local treatment of a serious burn. *A*, washing of burned area with a detergent soap and water *B*, irrigation with warm physiologic saline solution *C*, débridement of loose skin. Blisters need not be opened. *D*, application of a nonadherent dressing, such as petrolatum-impregnated gauze. *E*, application of plain gauze compresses *F*, application of sterile mechanic's waste or "fluffed" compresses The hand is then covered and immobilized in the "position of function" and an outer elastic roller bandage applied.

vent infection in third degree burns. Extensive cleansing and débridement are rarely necessary It is simpler and probably equally effective to use a detergent only when grease must be removed.

Petrolatum-impregnated gauze should first be applied to the burned area, and over this many sponges and finally a bulky material, such as sterile mechanic's waste. Over the entire dressing a circular elastic bandage should be evenly applied. Compression serves to reduce

eight to seventy-two hours, and if the burn bed is clean, it is usually ready to receive skin grafts. Red granulation tissue will not be present, but the raw surface will accept grafts and they will grow with a minimal scarring. If, instead, the surgeon awaits spontaneous separation of the slough and regeneration of epithelium, the course will be prolonged and complicated, and often the ultimate result will be unsatisfactory. It may take days, weeks or months before strands of collagen which bind the dermis to the subcutaneous tissues are digested and separated. In the meantime, infection inanition and "stalled" convalescence occurs. The earlier the area can be freed of slough and covered with skin, the less will be the scarring, deformity and mortality.

During this time, anemia may be a serious problem. If the patient has not received whole-blood transfusions as a part of his initial therapy, the anemia may be severe. It is necessary to check the hemogram and provide blood as necessary. The food intake is all important. Often it is necessary to augment the intake with formula feedings administered through an indwelling polyethylene nasogastric tube. (See Chapter 5, on Nutritional Balance.)

The patient should be returned to the operating room two or three days after débridement, for covering of the areas with skin grafts. The extent of the grafting procedure must be determined on the basis of the coverage needed and the patient's general condition. In severe burns, it may also be necessary to stage the skin-grafting operations The raw areas may bleed slightly when first exposed. The surrounding areas of intact skin should be washed with bland soap, or detergent, and water. The whole area should then be rinsed thoroughly with saline solution. If brisk bleeding occurs, the area should be temporarily covered with a compression bandage. Thrombin solution, applied locally, may aid hemostasis. Split-thickness grafts should then be removed from the donor area, usually by means of a dermatome, and applied to the recipient area. In general, the thinner the graft, the better the "take" (Fig. 33). Petrolatum-impregnated gauze dressings are applied to both the donor and the recipient areas.

When the burned patient has been permitted to go without skin coverage for long periods, the loss of protein, red blood cells and electrolytes from the granulating surface may be excessive. Patients in this state usually present an entirely different problem from that encountered in recently burned patients. Malnutrition, anemia and hypovolemia may be severe. Blood transfusions and careful dietary

(41° C.) cannot be tolerated for many hours. If less than 50 per cent of the skin surface is accessible for sponging with an alcohol and water mixture, it may be necessary to remove the dressings and expose the burned areas to the air. The room should be cooled and electric fans used to bring the patient's temperature down to more normal levels. If an air-conditioned room is available, this should be used Heat cradles should never be placed over burned patients.

It is known that patients with severe burns require large quantities of vitamin C During stress the adrenal cortex probably requires large amounts of ascorbic acid to maintain its function. Vitamin C is therefore given in high dosage parenterally during the treatment of the severely burned patient.

There is some danger in attempting to replace protein losses immediately by a high protein intake. A pseudo-diabetic state may result from the augmented excretion of adrenocortical hormones during the early postburn phase. There may be excessive fluid loss associated with glycosuria, severe depletion of the blood volume and shock. Accordingly, feedings which may aggravate the glycosuria are withheld in the early period of profound response to stress.

Because immediate assessment of the depth of a serious burn is difficult and usually inaccurate, final evaluation is generally deferred for ten to fourteen days. After this period, first and second degree burns will be healed, or nearly healed, and third degree burns will be evident. At this time, excision of necrotic tissue should be performed and preparations for grafting made Various substances have been suggested to speed separation of the necrotic tissue, but none have proved completely successful. The substances which have been tried include, pyruvic acid and proteolytic enzymes such as streptokinase and streptodornase, pancreatic trypsin and collagenase (an enzyme derived from Clostridium histolyticum).

When surgical excision of the slough is undertaken, adequate whole blood must be available for transfusion in case severe blood loss occurs. If a well-defined plane separates the dead and healthy tissues, blood loss is often slight, but this may not be anticipated in advance.

After the dead tissue has been removed, the raw surface is covered with fine-mesh petrolatum-impregnated gauze and pressure dressings to control oozing In extensive burns, débridement of only selected areas consistent with the safety of the patient may be desirable. The procedure then may be completed in stages. After a delay of forty-

the dressing. The whole area should again be covered with more gauze or sterile mechanic's waste and then secured with an elastic bandage. The irrigating fluid (usually saline solution) can be introduced at frequent intervals (usually four hours) in order to maintain a wet dressing. Under aseptic conditions the dressings should be changed daily.

As soon as the granulation tissue becomes clean, red and healthy-appearing, skin grafts should be applied. If the granulations appear unusually prominent (exuberant), they may be shaved down with a sharp skin-graft knife. All bleeding must be controlled before skin grafts are applied. The grafts are placed on the recipient area, generally without suturing, and fine-mesh petrolatum-impregnated gauze, sponges and elastic bandages reapplied. When grafts are placed over joint surfaces, splints or plaster casts are incorporated in the dressing to insure immobilization. In five or six days the grafted areas may be redressed.

The back is the preferred area from which to obtain skin. Here the dermis is thick, and healing occurs rapidly. If a very thin graft is taken, the donor area will usually heal in ten to fourteen days; and, if necessary, it may again be used as a source of skin for grafting. Unless infection develops, the donor area need not be redressed until healing is complete.

In extensive burns, it is occasionally necessary to apply *homografts* which will serve as a temporary covering during the critical early postburn period. This situation is met especially in children, and sufficient skin donor area may not be available. Homografts may be removed from healthy donors (without regard to blood type), or from cadavers immediately following death, and applied to the burned surfaces. If possible, islands of the patient's own skin (*autografts*) should be placed between the homografts. The homografts will disintegrate and disappear in six to eight weeks, but the autografts will provide a continuing source for re-epithelization.

Burns of the hands, joints and face require special attention. Burns of the hands must be débrided and covered with skin as soon as possible. The longer the delay in securing skin coverage, the greater will be the scarring and disability (Figs. 34 and 35). The hand should be held in the "position of function" while immobilized; and as soon as conditions permit, it should be mobilized. When skin is applied to burned areas over joints, the maximal surface area should be grafted and the part should be immobilized in a plaster splint.

management are necessary to reverse these deficits. As soon as the blood volume and red cell mass has been restored, and nutritional deficiencies have been corrected, rapid improvement will be observed.

The infected granulation tissue of neglected burned areas can be stimulated and cleansed with saline dressings. Immersion of the patient into a clean, warm bath, to which two or three handfuls of

FIG. 33.—The palm of the hand after application of a split-thickness skin graft for burns. Here a 100 per cent "take" of the graft is shown. Mobilization with a view to restoration of function is now possible.

salt is added, may be required in extensive burns. This helps clean up the areas, permits easy removal of dressings and permits early and painless movement of joints which are not covered with granulation tissue. In some instances when gross infection exists, a detergent or soapy solution may be added to the bath water. When there is a great deal of green pus (pyocyaneus), wet dressings containing ½–1 per cent acetic acid solution will be effective in suppressing the infection. The granulations are best covered with fine-mesh gauze, which permits wound drainage but does not allow granulation tissue to grow into the interstices of the gauze. Ordinary gauze sponges should be applied over the fine gauze, and irrigation catheters incorporated into

For example, in a burn over the extensor surface of the elbow, the skin graft should be applied with elbow in flexion, and a splint applied to hold it in this position. Similarly, if the burn is on the flexor surface, the graft is applied and the elbow immobilized in extension. For burns of the face, neck and axillae, early skin coverage is also required in order to avoid excessive scarring and contracture.

Many of the late complications of burns, such as contractures, deformities, and keloids, can be minimized by proper handling; but some are inevitable because of the magnitude of the original injury. Most burns are more than skin deep. Their correction comprises an interesting but complex facet of specialized surgical treatment.

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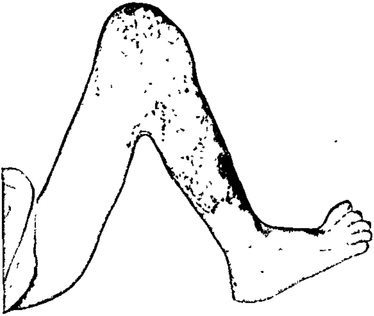

Fig. 34 *(above)*.—Late result of delayed healing of deep burns of the face and upper extremities in a child Note the marked scarring, flexion contractures (neck, elbows, wrists and hands) and keloids and the donor area on the lower chest from which a skin graft had been taken Some of these complications could have been prevented by earlier grafting and mobilization

Fig 35 *(below)*.—Delayed healing and marked contracture of the knee due to a neglected deep burn Treatment at this time consisted of immediate covering of the burned area with skin grafts and subsequent excision of scar tissue from the popliteal area and skin grafting with the knee in extension.

FIG 36.—Keloid formation in an old laceration of the shoulder. Note that the vaccination scar does not show this tendency to fibrous tissue hyperplasia.

strong case for hyperplasia as the underlying disturbance. However, the issue is principally of pathologic, rather than clinical, interest and need not concern the student at this time.

MALIGNANT TUMORS

The term "cancer" is used loosely to designate all types of malignant tumors. A malignant tumor is a new growth which exhibits a specific type of biologic behavior: if left untreated, it will ultimately bring about the destruction of the host by virtue of its excessive growth and dissemination. The outstanding characteristic of cancer is its biologic effectiveness. Cancer cells are set apart in that their ability to survive within the host is greater than that of normal cells. This ability is related to certain intracellular mechanisms which are transmitted in an unbroken sequence to all subsequent generations of cells. Once the intracellular changes appear, neither the original cell nor its progeny is able to revert to normal. By analogy, cancer cells closely resemble

CHAPTER 9

Basic Considerations in Management of Tumors

A TUMOR is defined as a swelling, a mass or a space-occupying lesion from any cause, but it has come to mean a new growth or neoplasm as a result of time-honored and wide usage. A neoplasm is a tumor derived from the tissues of the host. As such, it must be differentiated from malformations and masses due to hyperplasia, inflammation and repair phenomena.

All tissues of the body are able to produce new growths, but this ability is most often exhibited by those tissues having the greatest regenerative capacity and the least specialized function. New growths may be benign or malignant, according to their clinical behavior and pathologic characteristics.

To the clinician, a benign tumor is a neoplasm whose biologic behavior is such that the life of the patient is not endangered, except by reason of the size, position or complications which the tumor may cause locally. In contrast with malignant tumors, benign tumors remain confined to the area of origin and do not invade tissues or metastasize.

It is often difficult to distinguish between benign tumors and localized hyperplasia of tissues, for one may blend into the other. Hyperplasia can result from an exaggeration of tissue repair, such as is seen in keloid formation (Fig. 36); or it may result from endocrine dysfunction, as in hyperplasias of the breast and prostate. The problem of separating benign neoplasms from the hyperplasias is pointed up by such diverse conditions as: colloid goiter, leiomyoma of the uterus and functioning islet-cell tumors of the pancreas. One might logically question the existence of benign neoplasms as such and make a

The growth of cancer is characterized by excessive and accelerated cellular reproduction, local infiltration and destruction of normal tissues, and metastasis. "Metastasis" refers to the independent growth of tumor fragments which have become detached and transported elsewhere in the body. The tissue fragments may be disseminated by embolization, permeation or gravitational spread. The routes of transport are: the lymphatic channels (Fig. 37), the blood vascular channels, the cerebrospinal fluid system and the serous cavities. The pattern of infiltration and metastasis for a given neoplasm is predictable within certain limits. Lymphatic metastases usually occur sequentially to regional lymph nodes, then to more distant nodes and eventually to the venous system, through which widespread dissemination occurs. Blood-borne metastases are generally filtered out in the lungs and the liver, where they grow and produce other metastases. Blood-borne metastases from certain organs (breast, thyroid, prostate, kidney, lung) often lodge and grow in the bones of the axial skeleton. Presumably, tumor emboli may be conveyed to bone through the vertebral venous plexus without passing through the lungs (Batson).

Etiology of Cancer

Etiologically, cancer is a group of diseases rather than a single disease entity. The ultimate cause or causes of cancer are obscure, but many factors are known to be concerned with carcinogenesis.

Cancer occurs predominantly in the older members of the population, and the incidence of cancer increases greatly with aging. In infancy and up to five years of age there is a minor peak, which is followed by a decline during puberty and early adult life. The incidence increases at age thirty and continues to mount with advancing age. Initially the rate of increase is greater in females, but shortly it is surpassed in males. While this is the over-all general pattern, there are wide variations in the incidence of cancer according to specific sites and sex. The student is urged to consult works on the epidemiology of cancer for a consideration of these aspects of the cancer problem.

A genetic or hereditary predisposition to cancer is suggested by many experiments in animals. The role of heredity in humans has been established in such conditions as retinoblastoma, xeroderma pigmentosum and polyposis of the colon, but its relationship in other neoplastic diseases has not been established. The problem is understandably difficult to unravel, but progress is being made.

the very primitive forms of life in their ability to maintain an unbroken line through division of each cell.

The ability of the cancer cell to perpetuate itself is enhanced by the fact that it can thrive in most tissue and fluid environments of the body. Thus, cells which are transported to other areas can "take root" and form secondary tumors or metastases. Furthermore, cancer does not usually induce a defense reaction on the part of the body designed to destroy the invading cells. The relationship actually may

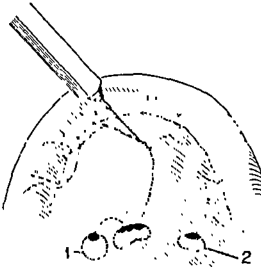

FIG 37.—Distribution of carbon particles after injection into a mesenteric lymphatic vessel Note that the particles pass directly to the first node, then to an adjacent node (*1*), then by other channels to nodes not in direct communication (*2*). The latter type of involvement may be considered an example of retrograde flow which occurs when the direct channels are occluded. This condition may occur when the lymphatics are blocked by inflammation or tumor cells It is believed that lymphatic dissemination of tumor emboli occurs in a similar manner. (From Gilchrist)

be one of tolerant coexistence until such time as the normal tissues are surrounded and destroyed. This is not to imply, however, that the body possesses no natural deterrents to the growth of cancer cells. That there must certainly be such mechanisms is indicated by frequent periods of quiescence, occasional instances of spontaneous regression and rare spontaneous cures.

Cancer cells also possess the ability to defend themselves. This is indicated by the fact that some tumors which are radiosensitive initially may become radioresistant later, and those which respond to certain chemotherapeutic drugs may later become nonresponsive.

Diagnosis of Cancer

The gross characteristics of cancers vary according to the tissue and site of origin, the growth potential of the tumor, the available blood supply, the local complications produced by the tumor (inflammation, ulceration, hemorrhage, necrosis, obstruction, etc.), the reaction evoked in the surrounding tissues and, to some extent, the age, sex and general condition of the patient (Fig. 38).

Many cancers tend to grow rapidly and to infiltrate widely. Cancerous tissues are usually more firm and less freely movable than normal tissues. Characteristically, neoplasms are painless and

Fig. 38 —Basal cell carcinoma near the eyelid in an elderly man The lesion was treated by irradiation.

insensitive because they possess no nerve tissue. Only as they compress, invade or otherwise irritate nerves do they produce pain and tenderness. This is the fundamental concept which von Mikulicz had in mind when he said: "There are no symptoms of cancer, only symptoms of the complications of cancer."

The microscopic appearance of cancer varies also, but generally the pathologic diagnosis is based upon well-defined criteria. These include: lack of normal cellular differentiation, bizarre cell forms, increased staining properties (hyperchromatism), abnormal numbers of mitotic figures, disorganization of the histologic pattern, abnormal cells beyond normal cell boundaries and abnormal cells within lymphatics, blood vessels or lymph nodes.

Biopsy is necessary for the definitive diagnosis of cancer. It is defined as the removal and examination of tissue from a living subject for diagnostic purposes (in contradistinction to necropsy). It is

A number of dietary factors have also been implicated in certain types of cancer. For example, it is postulated that the high incidence of gastric cancer among the Japanese may be related to a low intake of vitamin B.

Certain metazoal parasites, notably liver flukes, appear to incite hepatic cancer. Whether such tumors result from local irritation or specific carcinogens secreted by the parasite is unknown.

Chronic irritations, associated with repeated tissue destruction and repair, are prone to lead to malignant growth. This is exemplified by the development of cancer in long-standing skin ulcers, chronic sinuses and protracted ulcerative colitis.

Radiation and radiation injury also predispose to cancer. Gamma radiation and x-radiation, both valuable modalities in the treatment and control of neoplasms, may produce cancer by injudicious exposure. Similarly, excessive exposure to solar radiation may lead to skin cancer.

The polycyclic hydrocarbons derived from tar and petroleum are known carcinogens. Most of these substances produce cancer at the site where they are applied. The type of neoplasm which results appears to depend more upon the method of application of the carcinogen than upon the specific action of the substance. For example, if a certain substance is applied to the skin over a long period of time, a carcinoma will develop; if the same substance is injected, a sarcoma will appear.

The role of hormones in the initiation, growth and regression of certain neoplasms has received much attention. There is ample evidence to indicate that the estrogens are concerned with the growth of breast cancer, that androgens are associated with the growth of prostatic cancer; and that, experimentally, blockage of thyroid hormone production with massive doses of antithyroid drugs results in malignant goiter. The therapeutic value of hormones in advanced cancer is also well documented.

There is much to support the theory that cancer is caused by viruses. Tumors of virus origin are widespread in nature. They have been identified in plants and animals, but, as yet, have not been recognized in man. However, it is reasonable to believe that at least some forms do exist in man. Research in this field is progressing at an accelerated rate with the addition of new viruses and new diseases caused by viruses each year. It is possible that this work will one day bring an answer to the mystery of unrestrained cell growth.

With respect to the indications for and the conduct of biopsy, certain details should be kept in mind:

1. Pigmented moles should be removed completely, along with a margin of normal tissue. They should not be incised or cauterized.
2. Other small accessible lesions which might prove to be malignant should also be removed with a margin of normal tissue if possible.
3. Lymph nodes should not be biopsied until an intensive search has been made for a primary lesion. This rule applies particularly to enlarged cervical nodes, which often are the first sign of head and neck cancer.
4. The specimen submitted should include a margin of normal-appearing tissue if possible. In large ulcerating lesions, several pieces of tissue are usually removed (e.g., cancer of the rectum).
5. Frozen-section examination of the specimen should be obtained if there is need for an immediate pathologic diagnosis (e.g., during operation for breast tumor).
6. One must never accept a negative biopsy report as final so long as any suspicion remains that the lesion may be malignant. Repeated biopsies are often required to establish the true diagnosis.

Clinical Manifestations of Cancer

Malignant disease is characterized clinically by its insidious and silent onset. Generally, no significant symptoms or signs appear until the growth becomes large enough to produce a lump or to cause impaired function of some organ. As the process advances, it causes ulceration, bleeding, displacement, necrosis, infection or obstruction to hollow organs, which lead to observable clinical changes. During the early period of tumor growth, many times patients will recognize that there is something wrong with them but will ascribe their symptoms to vague disturbances such as "the flu," "run-down condition," "dysentery," "piles" or "misery." Such complaints appearing in a previously well person always bear investigation despite the fact that the patient regards them as insignificant and asks only that the physician "give me some medicine to fix me up."

The "seven danger signals of cancer" have been formulated and

the single most informative investigative measure for purposes of diagnosis, treatment and prognosis

In order that biopsy fulfil the purpose for which it is designed, the tissue sample submitted to the pathologist must meet certain requirements It must be large enough to work with and representative of the lesion from which it was secured. It must not be crushed, burned, dried out or otherwise distorted. Unless examined immediately, it must be placed in a fixative solution, usually Formalin. Each specimen bottle must be carefully labeled and submitted with a record of the patient's name, age, sex, the clinical diagnosis and a gross description of the lesion, the site from which the specimen was obtained and a brief résumé of the clinical history. All this information will be helpful to the pathologist

In the management of neoplastic disease it is a fundamental rule that, *before treatment is begun, the diagnosis must be established by histologic means, if possible.* This rule is based on the fact that experience has shown clinical diagnosis alone to be unreliable and frequently erroneous. The errors in clinical diagnosis fall into two general categories· failure to make the diagnosis of cancer because the lesion is thought to be benign; and to make the diagnosis of cancer when the lesion actually is benign Errors of this sort always do harm to the patient, to the physician and to medicine in general They are understandably the basis for many malpractice suits. It therefore behooves the physician, when dealing with tumors, to use every precaution, including biopsy, to protect the interests of all concerned

When the lesion is accessible, diagnostic biopsy may be performed as an integral part of the examination of the patient. Although there probably is some danger of spreading malignant cells into lymphatics or blood vessels when the sample is removed, the value of the examination outweighs the theoretical risks. It is essential, therefore, that the biopsy procedures be made as atraumatic and as bloodless as possible.

The tissue sample can be removed by incision of a part of the lesion, by excision of the entire lesion or by use of punch-biting forceps, a curette, an abrasive instrument, a suction or aspirating device or a simple swab. The manner in which the specimen is obtained is probably less important than that the sample be adequate in size and representative of the tumor. Crusts and necrotic debris are of no value in diagnosis

or completely unsuspected abnormalities. Generally speaking, the intelligent patient will not censure the physician when, after examination, the verdict is "no cancer"; but he will not forgive the physician who has belittled his complaints or failed to make an examination, when subsequent events prove he had cancer.

The physical findings in patients with early cancer in inaccessible sites are also likely to be within normal limits. Only when the disease

FIG. 40.—Advanced metastatic carcinoma in the lymph nodes of the neck from a primary tumor at the base of the tongue. A biopsy incision is present over the mass. The diagnosis could have been made more simply and probably more safely by locating the hidden primary tumor and securing a tissue specimen from it The patient was treated with radiation but succumbed later to ulceration, sloughing and hemorrhage from the neck.

becomes sufficiently advanced can detectable changes be expected. When the tumor is located in an accessible area, certain macroscopic tissue changes usually appear. They include: a mass which enlarges rapidly, a localized area of tissue thickening or induration, a chronic ulcer which fails to heal and bleeds easily or enlarged lymph nodes which lie in the lymphatic pathways of a previously undiscovered tumor.

The symptoms and signs of cancer of specific organs and tissues

publicized by the American Cancer Society. This listing, although admittedly incomplete, may be regarded as the clues upon which the search for the common types of cancer can be based. We believe the physician should know these seven signals and be in a position to teach them to others. They are:

1. Any sore that does not heal quickly, especially about the mouth (Fig. 39)
2. Any unusual bleeding or discharge from natural body openings

FIG 39.—Epidermoid (squamous cell) carcinoma of the lower lip. Note the exophytic nature of the growth The primary lesion was treated by irradiation; the cervical lymph nodes by bilateral suprahyoid dissection.

3. Any painless lump, especially of the breasts, lips, tongue or soft tissues
4. Any persistent indigestion or unexplained weight loss
5. Any progressive changes in color of a wart or birthmark
6. Any persistent hoarseness or cough or difficulty in swallowing
7. Any unexplained change in normal regular bowel habits

The symptoms of cancer are nonspecific and resemble those produced by many diseases of infectious, metabolic, nutritional, neurogenic and psychogenic origin. It is understandable that the diagnosis of cancer will often be missed unless the physician is trained to "think cancer" and is prepared to search for cancer on the basis of suspicion alone. In many instances the search will be negative for cancer. Under these circumstances, both the patient and the physician should be happy with the result. More often than one might expect, however, the search will turn up potential or actual malignant disease

The physician must also know the indications for and the limitations of special diagnostic procedures, including x-ray examination and endoscopy. Furthermore, he must consider the need for medical consultation whenever the problem is obscure or whenever he is not able to perform indicated diagnostic measures.

It is helpful to conceive of the search for cancer according to the accessibility of certain regions to examination. The American Cancer Society, in a booklet entitled *Cancer Detection in the Physician's Office* (1955), lists the following regions classified according to their accessibility to examination:

1. Visible: skin, lips, mouth, tongue, vulva, penis
2. Visible with instruments. vagina, anus, rectum, sigmoid, cervix uteri, urinary bladder, larynx, lungs, stomach, esophagus, nasopharynx
3. Palpable: breasts, rectum, prostate, ovaries, bones, testes
4. Inaccessible (except by x-ray examination): liver, intestines, stomach, pancreas, lungs, kidneys, brain, bone

In the same booklet, "The Seven Tragic Diagnostic Mistakes," which in a sense are corollaries of the "Seven Danger Signals," are outlined. These mistakes are:

1. Failure to make a diagnosis of intra-oral cancer on the assumption that it is a "canker sore" is an error that is avoided simply by making a biopsy of the lesion.
2. Failure to diagnose carcinoma of the breast because the physician believes the lump is benign, such as fat necrosis or inflammation, should never occur. A lump in the breast must be assumed to be malignant until biopsy proves it otherwise.
3. To treat a patient with the conviction that his symptoms are due to duodenal or benign gastric ulcer without radiologic or laboratory evidence of the disease is to miss the diagnosis of cancer of the stomach. Barium x-ray examination of the stomach, gastric analysis for acid content, and study of the feces for blood should be performed prior to beginning treatment of a suspected peptic ulcer.
4. Failure to recognize that an inguinal hernia, especially of long duration, which suddenly becomes symptomatic may be associated with carcinomatous lesions of the prostate or colon is a pitfall to be avoided. The dynamics of straining to void or defecate may be the cause of sudden increase of the symptoms from the hernia. In such instances the physician should investigate the possibility of prostatic or colon lesions and not devote all his thought to the hernia.
5. Tc [illegible] w [illegible] th [illegible] be given hormonal or other medication until cancer of the uterus has been excluded by adequate histologic examination.

are discussed elsewhere and will not be detailed here. However, the student should be able to synthesize the clinical picture which might be expected to appear in cancer of the different areas of the body on the basis of his knowledge of the manner in which tumors behave and the pathophysiologic changes which they produce.

The physician's search for cancer requires that he utilize all accepted investigative technics. Sometimes the clinical history will

FIG. 41.—Fibrosarcoma of the shoulder in a 9 year old boy. Radical local surgical excision resulted in cure.

provide a clue to the location of a lesion, but often it is of little help or may even be misleading. Frequently, too, the patient will have no complaints and simply wants a "checkup for cancer." Irrespective of the circumstances, nothing short of complete examination should be done (Fig. 40).

The survey must include special attention to inspection of the skin (Fig. 41), inspection and palpation of the body openings, and examination of the head and neck, breasts, chest, abdomen, anus, rectum and sigmoid, and the genitalia. The blood, urine and stool (for blood) should be examined, and an x-ray of the chest is usually in order. In females a cervical smear is made for study of the exfoliated cells. All patients who present suspicious accessible lesions should have them biopsied.

FIG. 42 —Cancer of the transverse colon. This specimen illustrates the extent of en bloc resection for cancer. Note that, while the lesion appears to be small, it contracts and obstructs the bowel. The lumen of the bowel above the tumor is dilated and the bowel wall is thickened (hypertrophied). Below the tumor, the bowel is of normal size and not thickened Several anatomic structures should also be observed: reflected above the transverse colon is the greater omentum; below and within the

ileum to the distal divided end of the colon. There were no metastases to the mesenteric lymph nodes. The patient made an uneventful recovery.

most forms of cancer are predisposed to spread to the regional lymph nodes before they become generalized. Therefore, the type of operation most likely to effect a cure is one in which the entire tumor and the regional lymph nodes which lie in the path of its lymphatic drainage are widely removed in continuity or en bloc (Fig. 42).

En bloc resection is applicable to many forms of cancer, but there are notable exceptions. Certain neoplasms do not spread through lymphatics (e.g., basal cell carcinoma of the skin); therefore regional lymph node dissection is unnecessary. Other tumors may be unsuitable for en bloc dissection for various reasons—for instance, when

6. Failure to recognize that bleeding piles may mask a coexisting rectal carcinoma is a tragic oversight. Thus, even in the presence of bleeding hemorrhoids, it is necessary to exclude the possibility of coexisting polyps or cancer of the rectum and colon, by digital rectal examination, proctosigmoidoscopy and barium enema.
7. To treat anemia without recognizing that cancer may be the primary cause of the blood loss is a serious error. Cancer anywhere in the body may be associated with anemia, and gastric and large bowel cancers are notorious for the anemia they cause.

Surgical Treatment of Cancer

Cancer is curable if it remains accessible to removal by operation or to destruction by irradiation. In most types of neoplastic disease the period of optimum curability is between onset of the first observable local changes and the first metastases. In some cancers, however, metastases develop almost at the onset of tumor growth, and in others they appear late in the disease. Thus, the duration of localized growth of cancer is extremely variable and is not simply a function of time It involves many other factors, such as age, sex, endocrine balance and heredity of the individual, as well as the type, location and inherent growth potential of the neoplasm. Much remains to be learned about these facets of tumor biology.

In general, the shorter the interval between the onset of tumor growth and the application of definitive treatment, the better will be the results of treatment. But it is well known that the exact extent of the cancer usually cannot be predicted on the basis of clinical findings alone, except when the patient presents unequivocal signs of incurability, such as distant metastases. For this reason, the surgeon is obliged to treat patients with "early" and "late" (but possibly curable) cancer alike. He cannot alter his treatment from one patient to another. Preoperatively, he must consider the possibility that the "early" cancer has metastasized and that the "late" cancer has remained localized. The treatment which he applies must be equally intensive and radical in both groups. When the disease has been demonstrated to be incurable, then all forms of radical treatment are contraindicated and probably harmful.

Surgical excision is currently the most important modality for the treatment of cancer. Surgical treatment is based on the premise that, if the entire malignant process can be removed from the body, then the disease will be cured. But local removal is not enough, because

infection, obstruction, distention, pleural and peritoneal effusion, etc., and *general treatment* for malnutrition, anemia, lowered blood volume, debility and emotional and mental disturbances. Special attention must be directed to the alleviation of pain, fear, worry, anxiety, insomnia, depression, suicidal tendencies and psychoses. Although the many important facets in the care of the patient with advanced cancer which warrant careful study cannot be discussed in this work, it is hoped that the student will pursue this subject (see Suggested Readings).

Operative measures which are designed to provide some degree of palliation are of several types:

1. Removal of the primary tumor, if possible, in selected cases
2. Short-circuiting procedures when the tumor is blocking a hollow organ and is irremovable
3. Nerve-cutting (neurosurgical) procedures for the relief of pain

Patients with removable but incurable cancer are often benefited by local excision of the primary growth. For example, some patients with ulcerating, smelly cancers of the breast with distant metastases are helped by removal of the breast (simple mastectomy). Such operations may provide a temporary but gratifying period of freedom from pain, bleeding, infection, etc., as well as improvement in the physical and emotional status.

Irremovable internal cancers which obstruct hollow organs are commonly by-passed or short-circuited According to the site of the obstruction, the by-pass may be internal or external with respect to the surface of the body. For example, cancers of the gastric outlet can be by-passed by making an anastomosis between the uninvolved upper portion of the stomach and the jejunum (gastrojejunostomy), or cancers of the right side of the colon can be by-passed by making an anastomosis between the terminal ileum and the transverse colon. In cancers of the lower part of the large bowel (rectum, sigmoid, colon) a communication can be made between the bowel proximal to the tumor and the abdominal wall (colostomy) to divert the fecal content away from the growth. Tumors which cannot be removed continue to grow and spread despite the by-pass, and the palliation achieved is often of short duration.

The neurosurgical measures for the control of pain in advanced cancer include: division of peripheral nerves, division of nerve roots (rhizotomy), division of pain pathways in the cord (cordotomy) and division of tracts of centers in the brain (tractotomy or lobotomy). The application of these procedures to patients with incurable disease

the primary tumor and the regional nodes are widely separated. Thus, a patient with melanoma of the foot would be treated by a discontinuous type of dissection; that is, the primary lesion and the lymph nodes in the groin would be removed through separate incisions and perhaps at different times.

It is a rule of cancer treatment that, when dissection is discontinuous either in time or space, the primary lesion must be eradicated first. If this order is reversed, the regional lymph nodes being destroyed first, the filtering function which they serve is lost and lymphatic emboli from the tumor are likely to pass to other nodes, which may be irremovable.

In summary, the principles of surgical treatment of cancer are:

1. Definitive surgical treatment should be applied only when the diagnosis has been established, the disease is removable with some hope of cure and the patient is physically capable of withstanding the proposed treatment.
2. The operation must be carefully planned on the basis of a knowledge of the natural history of the disease, the inherent anatomic and physiologic problems and the measures necessary for reconstitution of tissues after the resection.
3. The operation must be carefully executed with respect to the fundamentals of tissue handling, avoidance of contamination of the operative field with cancer cells and the general support of the patient.
4. The surgeon must make every effort to restore the patient to as near a normal state as possible. By its very nature, cancer treatment requires that some sacrifice of structure and function be made. How great the patient's sacrifice should be and how far the surgeon should go cannot be answered categorically. The surgeon may find the answer himself in the question: "What would I want done were I in the patient's shoes?"

Palliative Treatment of Cancer

The treatment of patients with incurable cancer is an important and often difficult aspect of medical practice The physician can bring relief in many ways to the patient and his family, and it may be said with respect to the true physician that this will be his "finest hour."

Measures available for the palliation of patients with advanced cancer include *local treatment* for pain, ulceration, sloughing, bleeding,

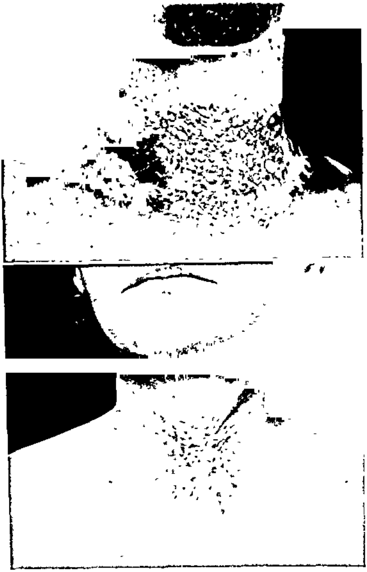

FIG. 43 *(above)*.—Early skin changes secondary to irradiation for cancer of the thyroid. Note the hyperpigmentation and scaling which resembles the healing stage in severe sunburn.

pointed out that external irradiation is no longer used in the treatment of hyperthyroidism.

requires careful consideration of the probable length of life; the type, location and severity of the pain; the effectiveness of other measures for pain control; the personality of the patient and his reaction to pain; *the chances that the neurosurgical procedure will be successful;* and the hazards and complications of these operations.

Local measures which are sometimes useful in patients with accessible but incurable lesions include: cleansing the ulcerated areas with liberal amounts of soap and water; saline soaks; irrigation of ulcerated and bleeding surfaces with solutions containing mild antiseptics, antibiotics or hemostatic substances; application of deodorant materials (especially activated zinc peroxide suspensions); and, rarely, application of topical anesthetic agents, electrocoagulation and ligation of large vessels entering the ulcerated area.

Radiation Treatment of Cancer

Cancer is also treated by exposure of the growth to ionizing radiation. Ionizing radiations include electromagnetic waves, such as x-rays and gamma rays, and high-energy particles, such as alpha and beta particles and protons. Ionization is described as the dissociation of individual atoms by which pairs of ions of opposite charge are formed by the dislodgment of electrons. The ionization lasts only a fraction of a second, but it results in chemical changes which lead to physiologic and morphologic effects which may be evident long after the exposure.

All normal cells can be destroyed by radiation, but susceptibility to its lethal effects varies greatly. Most sensitive are the lymphocytes, hemopoietic cells of the bone marrow, the germ cells of the reproductive organs and the lining epithelium of the alimentary canal. As a rule, sensitivity of a neoplasm to radiation is related to the sensitivity of the normal tissues from which the tumor originates. Thus, tumors which arise from cells mentioned above tend to be most radiosensitive. On the other hand, normal cells and new growths of bone, brain, muscle and connective tissue are less sensitive to radiation or more radioresistant. In cancer therapy, those tumors which arise from cells having a high mitotic activity and which are composed of adult cells *having a relatively short life-span respond best to irradiation.*

The injury produced by ionizing energy resembles that produced by sunburn but is more severe and deeper (Fig. 43). The early changes consist of capillary dilatation, edema, swelling of collagen and elastic

the palliative treatment of selected patients with specific types of neoplastic disease:

1. *Nitrogen mustard-type compounds*—nitrogen mustard, triethylene melamine (TEM), triethylene phosphoramide (TEPA) and triethylene thiophosphoramide (thio-TEPA)
2. *Steroid hormones*—androgens, estrogens, adrenocortical hormones and adrenocorticotropic hormone
3. *Antimetabolites*—amethopterin, aminopterin, 6-mercaptopurine (6-MP), azaserine

Psychologic Aspects of Cancer

The management of the psychologic and emotional disturbances encountered in patients with cancer is as much a part of the over-all treatment as is, for example, the maintenance of nutrition. Some problems in this area always arise when a person becomes ill or undergoes an operation, or fails to get well, or learns that he has cancer. Since this is the case, the physician must anticipate these reactions and take steps which are necessary for their amelioration. He must do this before such reactions become so serious as to jeopardize recovery or hasten the end. The key to success in this regard is a broad understanding of the psychology of illness and an intimate knowledge of, and personal feeling for, the patient as a person.

In order that the physician may best guide his patient through this difficult period, he must assume that most people have little knowledge regarding medical matters. Generally the patient's fear is increased by ignorance and decreased by understanding. The physician can allay fear by explaining the situation in terms which the patient can understand. He must, therefore, take the time to *listen to the patient* and to *talk to the patient* Furthermore, he must not take away "the last ray of hope" to which the patient clings; for when this is gone, there is nothing left for the patient to look forward to.

There is no unanimity of opinion regarding "what to tell the patient," and there appears to be no simple answer to this problem. Some physicians tell the patient nothing and try to protect him from the truth, others insist that the patient must be told the whole truth as gently as possible. But between these two extremes there are those who believe the answer should fit the needs of the patient himself. Thus, the type and extent of the patient's disease; the outlook for cure, prolonged invalidism or death, his understanding and equanimity; his

tissue, and damage to vascular endothelium. Edema and inflammation are regularly observed. Possible late changes are shown in Figure 44.

After exposure to small doses of radiation the tissues usually return to normal. With larger doses, there is loss of hair and atrophy of the skin appendages. Intensive irradiation leads to pigmentation, telangiectasis, permanent tissue swelling, induration and sometimes chronic ulceration, radionecrosis and occasionally neoplastic growth.

The systemic effects of irradiation include: anemia, leukopenia, radiation sickness and acute and chronic forms of the radiation syndrome.

The arrest or control of cancer by means of ionizing radiation depends on the amount of energy absorbed by the tumor. The particular form and dosage prescribed will vary according to the circumstances, but the biologic changes which result from all forms of radiation are quite similar qualitatively. The objective of all such treatment is to deliver sufficient radiation to eradicate or inhibit the tumor without producing undue damage to normal neighboring structures

Radiation therapy may constitute definitive treatment for certain radiosensitive neoplasms. It must be applied with the same care required in the surgical treatment of cancer. Currently, it is the treatment of choice in some clinics for many specific types of cancer, including cancer of the lip, cancer of the cervix uteri, some cancers of the skin and the lymphomas.

Radiation therapy is also widely used in the palliative treatment of malignant diseases for the control of neoplastic growth and the alleviation of pain. It can be applied through external irradiation (x-ray), through local application of radium or radioactive isotopes or systemically through the circulation in the form of radioactive isotopes

Other Forms of Treatment

Other useful measures in patients with advanced cancer include hormonal alteration and chemotherapy. Hormonal alteration is discussed in Chapter 27, on the Breast.

The search for effective chemotherapeutic agents for cancer is a continuing one. Innumerable substances have been investigated, and a few have been found which possess anticancer properties. As yet, however, none have been discovered which actually cure cancer.

Three classes of chemotherapeutic agents are now being used in

the palliative treatment of selected patients with [illegible] of neoplastic disease:

1. *Nitrogen mustard-type compounds*–nitrogen [illegible] ene melamine (TEM), triethylene [illegible] triethylene thiophosphoramide (thio-TEPA)
2. *Steroid hormones*–androgens, estrogens, [illegible] mones and adrenocorticotropic hormone
3. *Antimetabolites*–amethopterin, aminopterin, [illegible] (6-MP), azaserine

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responsibilities and obligations; his desires and those of his family; and finally, his attitude, as expressed by the questions which he asks, must be considered.

Some patients want to know nothing about their illness because they suspect they have cancer and do not want their suspicions verified. Others really would like to know but are afraid to ask the physician because they fear the news will be bad. Some patients, wanting reassurance, ask: "I don't have cancer, do I doctor?" And others will say: "I want you to tell me the truth, Doctor, do I have cancer?"

It would seem reasonable to believe that those who do not want to know need not be told; those who want to know should be told; those in between must be dealt with according to the circumstances and the surgeon's best judgment. If the patient is not told the true nature of his disease, it is essential that a responsible member of his family should be so informed. Generally, if the disease is incurable, one member of the family should be informed regardless of whether the patient understands his condition or not.

An important related problem is that of prognosis. The unpredictability of duration of life in patients with incurable cancer makes prognostication difficult. The physician should not attempt to predict that the patient will live any specific period of time, such as so many days, weeks, months or years. If duration of life *must* be estimated, it is better to give a time range which might include minimum and maximum estimates. Thus the prognosis may be estimated to be from three to six months, from six to twelve months, from one to two years, etc. Under these circumstances, all concerned—the patient, his family and the physician—will be better satisfied.

BENIGN TUMORS

Benign tumors and cysts of the body surface are usually easily diagnosed and simply treated. They are encountered at all ages, in both sexes, and most commonly on the exposed areas of the body. When searched for, however, such lesions are often found in less conspicuous areas. Since usually they cause no symptoms, they are not often the cause for concern on the part of the patient. However, benign tumors, as well as malignant tumors, are manifestations of abnormal growth, and the physician should consider the need for their removal. In this regard, he must recognize the frequency of errors in

clinical diagnosis and *must* submit all excised tissue for pathologic diagnosis, except when the macroscopic features clearly indicate the benign nature of the lesion (e.g., sebaceous cyst, lipoma, etc.).

When the clinical examination suggests that the lesion is probably benign but that it possibly may be malignant, the lesion should, if possible, be completely excised together with a rim of normal tissue. Frequent errors in this regard are: the incomplete removal of a mole which on histologic examination proves to be a melanoma, and the incomplete removal of a subcutaneous tumor thought to be a cyst or fibroma which proves to be a soft-tissue sarcoma.

Benign tumors tend to grow more slowly than malignant tumors and are usually encapsulated, localized and movable. They are less likely to undergo ulceration or necrosis and are usually asymptomatic. Some benign lesions are thought to be forerunners of cancer, and their removal is essential to cancer prophylaxis. The most common premalignant lesions are: leukoplakia, senile keratoses, certain pigmented moles, solitary thyroid nodules, adenomatous polyps of the rectum and colon and certain hyperplastic nodules of the breast.

Sebaceous Cyst (Wen)

These lesions occur most commonly on the scalp, face, neck and back, but they may appear anywhere on the skin They are the result of plugging of the normal sebaceous duct orifice, producing stasis of sebum in the gland. The gland gradually becomes distended with secretion and is thereby predisposed to infection with skin organisms. The site of the plugged duct orifice is often indicated by a prominent comedo (blackhead), which is usually found near the center of the dome of the cyst.

Sebaceous cysts are round, smooth, sometimes fluctuant masses located in the subcutaneous skin layer but attached to the epidermis. They are slightly movable and nontender, except when infected. The thin cyst wall encloses a white or grayish malodorous caseous material. The cyst is easily separated from the surrounding tissue except when it is fixed by scar tissue or inflammatory exudate. When the cyst is acutely inflamed, excision should be postponed until the reaction has subsided. Occasionally, simple incision and drainage is necessary to control infection. In most instances, sebaceous cysts are removed under local anesthesia and the incisions are closed primarily.

Mole or Pigmented Nevus

These lesions are the most common tumors of the skin. The average adult is said to have at least twenty benign nevi. They are rarely present at birth, but begin to appear during infancy and continue to increase in size and number at puberty. They vary from tiny flat brown spots to hand-sized elevated, hairy, warty, brown or black patches. They are most commonly located on the face, neck, upper extremities and upper trunk, but they occur on all cutaneous surfaces. Clinically, the importance of nevi lies in the fact that certain types are the forerunners of malignant melanoma. Melanomas have been reported to arise from pre-existing nevi in from 18 to 65 per cent of cases, and melanomas constitute only 1–2 per cent of all malignant tumors. Thus, the percentage of moles which become melanomas is very low. Their innocent appearance and frequent vicious behavior, however, require that they be given special attention.

Pigment-cell tumors are classified according to their clinical and histologic features, as follows:

1. *Junctional nevi*—flat or slightly raised, dark brown or black, and usually hairless moles. Nevus cells are found in the basal layers of the skin close to the dermal-epidermal junction. Junctional nevi are considered precursors of melanoma.
2. *Compound nevi*—comprising about 98 per cent of the nevi which appear before puberty and about 12 per cent of those seen in the adult. They may be brown, black or nonpigmented. They are occasionally pedunculated and may or may not contain hair. Nests of nevus cells are seen at the dermal-epidermal junction, as well as in the dermis. Those with junctional changes are considered precursors of melanoma
3. *Dermal nevi*—similar in appearance to the compound nevi. Nevus cells are found only in the dermis. Dermal nevi are not implicated in the development of melanoma
4. *Blue nevi*—steely-blue, isolated, hairless lesions which range from 1 to 3 cm. in size Melanocytes are confined entirely to the dermis, there being no epidermal or junctional involvement. These moles are benign but are often confused with melanomas.

Pigmented moles rarely become malignant before puberty despite the fact that they contain nevus cells at the dermal-epidermal junction and histologically are similar to melanomas. They have been called "juvenile melanomas" and may be the basis for true melanomas of

later life. Suspicious moles should be removed and submitted for microscopic study. The age of the patient must be made known to the pathologist in order that he will be able to make the diagnosis in light of the known behavior of these lesions.

Removal is indicated for pigmented tumors of the following types:

1. Any mole which spontaneously bleeds or ulcerates
2. Any mole which increases in size
3. Any mole with irregular borders, finger-like projections or accompanying satellites
4. Any mole so located as to be subjected to repeated trauma (belt line, sole, palm or fingers, genitalia, etc.)
5. Any mole which is brown-black or black, or shows an increase in the degree of pigmentation
6. Any mole of recent onset

The treatment of pigmented moles is surgical excision, including an adequate margin of normal tissue. Cauterization, fulguration, incision biopsy or other incomplete measures are to be condemned. The excised tissue should *always* be submitted for pathologic examination.

Melanoma

Melanomas are *malignant tumors* which arise from nevus cells. Opinion regarding the origin of nevus cells is divided between those who believe they are derived from primitive nerve cells and those who believe they are derived from ectodermal cells. Nevus cells may or may not contain pigment and, accordingly, the tumors which they produce may be pigmented or nonpigmented. Melanomas which arise from pre-existing moles develop only from those having a junctional component, but some melanomas develop de novo in the absence of a pre-existing mole.

The sites of predilection are: the lower extremities (especially the feet), the head and neck, the genitalia and the body orifices. They occur with equal frequency in men and women and are seen predominantly after age 30 The development of melanomas is often associated with some form of chronic injury or irritation, as, for example, when a mole located on the face is subjected to repeated injury from shaving.

Melanomas may be black, brown or flesh colored. Usually they have a slightly raised, irregular surface which is devoid of hair. When they become active, enlargement, ulceration, crusting and slight bleeding are common. As spread occurs, the area surrounding the

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LIPOMA

This tumor commonly appears in the subcutaneous areas of the extremities and trunk. It is the result of a localized increase in fatty tissue. Typically, lipomas are smooth, lobulated, soft, fluctuant (fat at body temperature is liquid), encapsulated masses (Fig. 46). They vary

FIG. 46.—Large lipoma of the axilla in a young woman The tumor was lobulated and soft. Note the unusually prominent venous pattern of the left side of the chest This is an infra-red photograph, which makes the superficial veins stand out.

markedly in size and grow slowly and silently. Except for their size, appearance and the fact that they occasionally become malignant (liposarcoma), they cause little difficulty. Sometimes they develop in the abdomen or chest, where they may become very large and, by compression or displacement, cause symptoms.

Lipomas are recognized by their location, discreteness, lobulation and consistency. They are cured by simple excision.

ANGIOMA

Angiomas are vascular defects which probably result from abnormal proliferations of embryologic arterial or venous structures.

primary lesion may become studded with pigmented metastatic nodules (satellites) (Fig. 45), and the regional lymph nodes may also become enlarged. Metastasis occurs via lymphatic and vascular routes to the liver, the lungs, the bone marrow and eventually to all parts of the body in the disseminating stage of the disease.

Complete surgical excision while the lesion remains localized is the only effective method of treatment. The local excision must be

Fig 45.—Satellite nodules of malignant melanoma in a 60 year old man. He had had a pigmented mole over
It suddenly became larger
this photograph was taken,
tissue was taken for examination. Later, regional and generalized metastases occurred The patient died of widespread dissemination.

wide and deep. Skin grafting is often necessary to close the defect. Amputations are sometimes required. Dissection of the regional lymph nodes is also in order. Radiation therapy is generally ineffective.

Wart (Verruca Vulgaris)

A wart is a common benign papillary tumor of the epidermis which occurs predominantly in childhood but occasionally in later life. Warts may appear in crops, and they often disappear spontaneously. The lesion is a raised, cauliflower-like projection which ranges in diameter from 1 mm. to 1 cm. Its surface is sometimes stippled with small dark dots. When traumatized, warts often ulcerate and bleed.

Common warts are treated by excision or fulguration. Unless they are offensive or subject to injury, it is probably better to leave them alone with the expectation that they will disappear.

the regulation of body temperature. Glomus tumors are most commonly located in the upper extremities (fingers, hand, forearm) but may occur elsewhere on the body surface. They are bluish or purplish, round, soft encapsulated nodules which vary from pinhead- to pea-sized masses. Characteristically, glomus tumors produce throbbing, burning or shooting pains. They are extremely sensitive to slight pressure or any irritation. The patient usually protects the sensitive area from all injury. Removal of the tumor results in cure.

Lymphangioma

This tumor results from an abnormal growth of primitive lymphatic tissue. The mass produced is soft, lobulated, fluctuant, nontender and often translucent. It occurs principally in the neck, axilla and groin of infants or children. Usually the skin over the mass is unchanged. *Cystic hygroma collicum* is the common form of lymphangioma of the neck in infants.

Lymphangiomas are treated by surgical excision. Those which are extensive and infiltrate the cervical or axillary neurovascular structures may require irradiation or injection of sclerosing solutions.

Senile Keratosis

This is a precancerous condition which develops on the exposed areas of the skin (nose, ears, cheeks, neck and dorsum of the hand) in fair-skinned persons past middle age. The lesions are slightly elevated, flat, flesh to yellow or black in color and adherent to the underlying skin They may contain horny surface projections, and they are associated with other skin changes of aging. Microscopically, there is thickening of the stratum corneum as well as atrophy of the skin and some inflammation. Senile keratoses should be removed by excision or electrosurgery.

Leukoplakia

Leukoplakia is also a precancerous condition. It appears as a whitish thickening of the mucous membrane, especially in the mouth, and the lesion resembles an adherent patch of white paint. It usually develops in men and is often associated with some form of chronic irritation (ill-fitting dentures, tobacco chewing, dental caries, etc.).

They may develop in any organ or tissue but are most often encountered in the skin and subcutaneous tissue. During infancy and childhood, small capillary hemangiomas often appear and then regress and disappear if left undisturbed. Large, conspicuous or expanding hemangiomas, on the other hand, may require treatment.

The common capillary hemangiomas are soft, pink, red or purple, flat birthmarks which vary widely in size, shape and coloration. They

FIG. 47.—Hemangioma of the nose in a child. Note the profusion of small venules or capillaries in the lesion The condition was treated by irradiation.

are treated by excision, irradiation or application of carbon dioxide snow

Cavernous hemangiomas occur in the subcutaneous or submucous tissues and are irregular, soft, reddish-blue compressible tumors They are composed of a profusion of dilated veins (Fig. 47), which sometimes communicate directly with arteries Under these conditions they may pulsate. When large arteriovenous communications are present, the lesion is called a *cirsoid aneurysm* Removal may be indicated for cosmetic or other reasons. Cavernous hemangiomas rarely undergo spontaneous regression.

Glomus Tumor

This is a rare benign tumor of the neuromyoarterial glomus of the skin. The normal glomus is a tiny conglomeration of arteriovenous communications containing nerve elements which is concerned with

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Leukoplakia progresses slowly and insidiously and tends to be resistant to all topical medication. It is treated by elimination of all local irritation and, if necessary, by excision or electrocoagulation.

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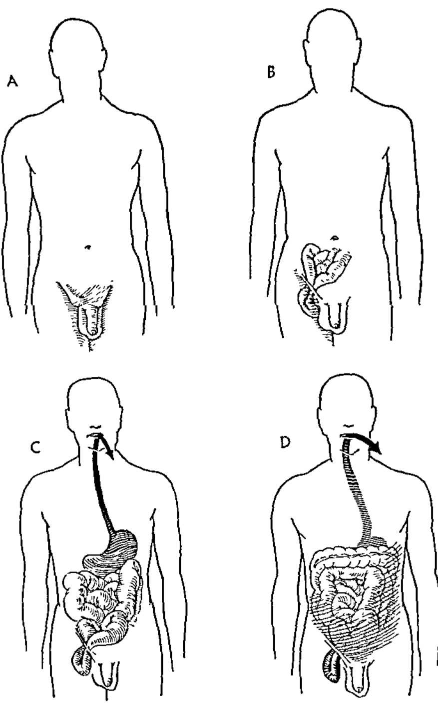

FIG. 48 —The surgical risk, illustrated by diagrams of inguinal hernia modified according to local and systemic disturbances as influenced by *time*. A, simple in- [illegible]

the gut and gangrene of the strangulated loop. Extremely poor surgical risk.

CHAPTER 10

Principles of Surgical Care

BECAUSE THE SURGEON is responsible for the *total* care of his patients, his responsibility extends beyond diagnosis and the operative technics which make use of the scalpel, hemostat and ligature. To lose sight of the patient as an individual in the abstractions of pathology or the intricacies of surgical technic is a tragic error. We as physicians deal with human beings—not a chronic gallbladder, a hernia or an interesting gastric cancer. Commenting on this concept of the *Total Care of Patients*, David Barr says:

> We who have received our training in the past have had our attention focused upon the recognition and treatment of organic and mechanical abnormalities. We have been obsessed with the fear of missing the presence of serious or potentially serious anatomical or chemical disease. Intellectually we have been occupied with the relationship of clinical signs and anatomical deviations, with the correction of chemical defects and with the search and application of specifics. We have been burdened with a time-consuming and elaborate ritual to accomplish these purposes. While all of this is praiseworthy, it is not enough if in the process we have lost the listening ear and our contacts with patients as people, or if we attempt to make diagnoses and decisions without consideration for personal problems, interpersonal relationships, and life situations in family, occupation and community.*

The patient who consults with a surgeon, for the first time, approaches the interview as a new and a strange experience. He has undoubtedly heard much about the unpleasant aspects of operative treatment. He anticipates that it will be both dangerous and costly. He may realize the importance of early diagnosis and treatment, yet prefer to delay as long as possible in the face of such an ordeal. All of this is understandable. He has countless worries and fears: Is he

* Barr, D. P., Ann Int Med (27.195, August, 1947)

life is critical and easily upset. The immature organism is predominantly unstable—sensitive to exposure, chilling, trauma, prolonged operation, rough handling and loss of blood and other body fluids. The loss of an ounce of blood in an infant is said to be equivalent to loss of a pint in the adult. Despite these limitations, it is possible in this age group, by careful planning, gentleness and precision, to apply surgical treatment with the expectation of a good response.

Advanced Age

The principles of treatment are no different for the geriatric patient. Often there appears to be a gross discrepancy between the chronologic and physiologic or biologic age of the patient, which may increase or decrease, as the case may be, the response to stress. In general, however, the degenerative changes of the aging process parallel the patient's years, and response tends to be less favorable than in the young. Nutritional deficiencies, emotional disturbances and difficulties in mobilization are only a few of the common disturbances encountered in advanced age. These factors, as well as others, decrease the patient's ability to adapt to the stresses of anesthesia and operation

When elective surgical treatment is being considered, the possible benefits which may be gained must be weighed against the possible risks. On this basis, certain operations for conditions which do not threaten life are not warranted. On the other hand, if an operation carries little risk and will add to the patient's comfort, activity or enjoyment of life (e.g., repair of an inguinal hernia), it is often justified.

In the aged person, special care must be given to avoid pulmonary and cardiovascular complications, thromboembolic disease, pressure sores and psychic disturbances (e.g., depression, disorientation).

Obesity

It has been said that "as we lengthen the waist line, we shorten the life line"; and certainly, marked overweight is always a cause for worry on the part of the surgeon. The very fat person does not stand the stress of surgery well, technical and mechanical problems are increased, wounds heal poorly and are easily infected, nursing care is more difficult, and mobilization of the patient sometimes almost impossible.

doing the right thing? Has he chosen the right surgeon? How will his family get along during his hospitalization? Will he be able to work after the operation? How much will it cost? Will there be much pain? Will he live or die? And so on.

The good surgeon must anticipate such questions and allay the patient's fears. He must aid the patient in the emotional adjustment to this new life situation. He must take time to talk to him in language which can be understood. Not only this, but the surgeon must make all decisions regarding diagnosis and definitive treatment; he must evaluate the operative risk (Fig. 48) and the possibility of complications, and, finally, he must make a prognosis. In addition, he must supervise the details of hospitalization, anesthesia, operative schedules, special nursing and convalescent care and, ultimately, the rehabilitation of the patient.

To the "busy surgeon," the infinite number of details may seem time consuming and often trivial; but on such considerations rests the dignity of the art of medicine, the success or failure of the patient-doctor relationship and, sometimes, the final outcome of treatment.

FACTORS INFLUENCING THE RESPONSE TO SURGERY

There is usually time to evaluate and treat those conditions which predispose the patient to complications and increase the risk of operation. Time expended to this end will generally pay high dividends in terms of improved surgical risk, smoother convalescence and quicker recovery

Many factors adversely influence the surgical risk; these include: hemorrhage and shock, dehydration and electrolyte imbalance, acidosis and alkalosis, anemia and hypovolemia, malnutrition and hypoproteinemia, pulmonary and cardiovascular disturbances, renal and adrenal disease, hepatic insufficiency, hyperthyroidism and diabetes, obesity, pregnancy, psychic and emotional disturbances, alcoholism and drug intoxication, and the extremes of age (very young and very old). Most of these conditions are considered in other sections; a few are described here briefly.

Early Life

In general, children withstand trauma well and exhibit remarkable recuperative powers even though metabolic balance at this stage of

Diabetes Mellitus

In general, the surgical risk of the patient with controlled diabetes does not exceed that of the nondiabetic. Sometimes the surgical condition (e.g., infection, gangrene of the extremity or acute cholecystitis) aggravates the diabetes and makes control more difficult. Conversely, uncontrolled diabetes appears to enhance the severity of the surgical condition. Combined problems of this type demand the exercise of critical judgment in timing surgical treatment.

Wound healing in the controlled diabetic is not delayed beyond that which can be ascribed to existing vascular insufficiency, usually from associated atherosclerosis. In uncontrolled diabetes, however, there exists an increased susceptibility to infection which is not well understood. It may be related to high local concentrations of carbohydrate and protein breakdown products associated with gluconeogenesis.

Patients in diabetic acidosis admitted for surgical treatment must receive prompt treatment for metabolic imbalance before being submitted to operation. In some cases, it will be impossible to completely control the systemic disturbance before the local problem has been improved or corrected (e.g, wet gangrene of the extremity, treated by primary amputation or by refrigeration with subsequent amputation).

Of special importance in the diabetic patient is the care of the skin, particularly the skin of the feet. The utmost cleanliness and the avoidance of cuts, abrasions and infections, which predispose to gangrene, are essential. The care of the feet is described in Chapter 28, on Peripheral Vascular Diseases.

The following instructions regarding the care of the diabetic patient have been reproduced, with slight modifications, from the *Diabetes Guidebook for the Physician*, published by the American Diabetes Association, Inc., in 1950.

Preparation of the Diabetic Patient for Operation

1. *Diet.*—The usual diabetic diet should be given in most cases. When there has been poor nutrition, give additional protein and vitamin supplements. When diabetes is complicated by hepatic disease, give more carbohydrate and protein; and when hyperthyroidism is present, give extra food to meet the increased caloric need.
2. *Insulin.*—Give insulin as needed to control glycosuria: protamine zinc insulin in a single dose before breakfast and, when necessary, supplementary doses of unmodified insulin before breakfast, occasionally before the evening meal as well.

Hypertension, myocardial disease, diabetes and hepatic and biliary disease are common in obese patients. The load imposed by operation may exceed the reserve capacity of the cardiovascular system. The increased cardiac strain in very obese patients is suggested by the estimate that for each 30 pounds of excess weight about 25 extra miles of blood vessels are needed. The increased heart load is obvious.

Fat tissues often unite poorly. Wound healing may be complicated by hematomas, serum collections, infections, and superficial or deep separation. After abdominal operations, ventral hernias are common. Both atelectasis and phlebothrombosis occur with increased frequency. Anesthetic problems are accentuated.

It is good judgment to insist that excessively overweight patients undergo weight reduction before elective operations are undertaken. This can be accomplished by adherence to a strict low-calorie diet (usually 1,000 calories) and close supervision by the physician while the patient is on this regimen. The amount of weight loss desirable is an individual problem. Once weight reduction has been accomplished, the outlook for an uncomplicated convalescence is improved.

Cardiovascular Disease

All patients who are to be subjected to anesthesia and operation must be examined regarding the functional capacity of the cardiovascular system. If evidence of acute or chronic heart failure exists, immediate operation is contraindicated except under emergency conditions. Operation should be deferred until maximal benefits have been obtained from medical treatment When emergency operation is necessary because life is threatened, immediate supportive measures (digitalization, oxygen therapy, transfusion, etc) should be instituted before and continued after operation.

Essential hypertension, compensated heart disease, a history of angina pectoris and some cardiac arrhythmias predispose the patient to cardiovascular complications but, by themselves, do not contraindicate operation. Patients exhibiting these disturbances require more than usual diligence on the part of the physician during all phases of management.

Items of critical importance include· avoidance of hypotension and hypoxia, avoidance of unphysiologic positions and sudden changes in position during operation, avoidance of prolonged immobilization, and avoidance of fluid and blood "overloads."

4. *Laboratory Tests.*—Test every specimen of urine for sugar; try to get specimens at least four times a day.

   When diabetes is severe, order a blood sugar test (capillary, if available) at noon and at 4 p.m. the day of the major operation—at 4 p.m. only when the operation is minor. Order additional tests at any time if there is any doubt as to the patient's diabetic status.
5. *Hypoglycemic Reactions.*—Think of an insulin reaction when any unusual complaints or behavior is noted. In cases of doubt, give treatment for hypoglycemia: 5–20 Gm. of sugar by mouth or by vein or 0.5 cc. of epinephrine subcutaneously.

Pulmonary and Upper Respiratory Disease

An existing acute respiratory infection generally contraindicates elective operation. Chronic disease of the lungs or air passages also increases the danger of postoperative pulmonary complications. Oral and nasopharyngeal sepsis similarly influence the development of complications. During the preoperative period, efforts should be directed toward decreasing sepsis and improving ventilation in all portions of the respiratory tract. This subject is discussed in more detail in Chapter 12, on Postoperative Complications.

Ventilatory disturbances, of any type, which impair uptake of oxygen and output of carbon dioxide lead to hypoxia and hypercapnia, with resultant serious metabolic imbalances. The need for maintaining adequate ventilation during all phases of treatment, and especially during anesthesia, is apparent.

Renal Disease

The existence of acute nephritis, acute renal insufficiency with oliguria or anuria, or other acute renal disturbances, contraindicates operation except under emergency conditions or when the operation is needed to improve renal function (e.g., prostatism). Renal function should be assessed by urinalysis, blood urea nitrogen and creatinine determinations, concentration and dilution tests and excretory tests, including excretory urography if necessary. Maximal improvement in renal function should be attained before elective operations are undertaken. The anesthetic agents most commonly used do not appear to influence renal function significantly.

Alcoholism

Patients who are acutely intoxicated often suffer injuries which require surgical treatment. Except when the injury is of minor degree

3. *Laboratory Tests.*—Absence of glycosuria and reduction of the fasting blood sugar below 160 mg. are desired but are not essential. Sugar in the urine during part of the day or high blood sugar need not delay an operation unless ketosis is present. On the other hand, there should not be haste in undertaking an elective operation if the patient's condition is poor because of previous neglect of diabetic treatment.

PREOPERATIVE TREATMENT

1. *Insulin.*—Give the usual dose of insulin (as previously used) the morning of operation. In addition, give unmodified insulin
   —if the diabetes is severe (as shown by requirement of more than 30 units of insulin daily),
   —[illegible]
   —[illegible]
   [illegible] lycemia in the operating room—not more than 5 or 10 units, unless nourishment is given by mouth or dextrose is given by intravenous infusion.

   To guard further against hypoglycemia in the operating room, consider reduction of the dosage of long-acting or intermediate-reacting insulin given before supper on the day preceding the operation.
2. *Nourishment.*—Before operation, nourishment is desirable if the diabetes is very severe or if it is complicated by hyperthyroidism.

   To give food by mouth is usually contraindicated unless four or more hours elapse before operation. An intravenous infusion containing 50–100 Gm. of dextrose is preferable. At the same time, an extra 10 units of unmodified insulin for each 50 Gm. of dextrose should be given intravenously.

POSTOPERATIVE TREATMENT

1. *Intravenous Infusions.*—Give fluids as would be indicated in the uncomplicated surgical case. If food cannot be taken by mouth, give 1,000–2,000 cc. of 5 or 10 per cent dextrose—to make the total intake of dextrose at least 125–150 Gm. in twenty-four hours.

   Give an extra 10 units of unmodified insulin subcutaneously for each 50 Gm. of dextrose given intravenously.
2. *Diet.*—When the patient is able to take nourishment by mouth, give liquids and soft foods. If necessary, give supplementary feeding with intravenous infusions of dextrose to bring the total carbohydrate intake up to 150 Gm After a minor operation, the patient may be given his usual diet immediately.
3. *Insulin.*—Give the usual morning dose of insulin if it was employed before operation. Give extra doses of unmodified insulin at mealtime, [illegible] ous in ordering [illegible] her than more, than may seem to be needed.

   A red test may show the need for 20 units of insulin in the morning, but with such a test in the evening 5 units may be too much if a large total dosage has been given earlier.

   Disregard glycosuria observed within 2 hours after an intravenous infusion of dextrose.

adrenal hemorrhage (Waterhouse-Friderichsen syndrome), severe burns or injuries. The deficiency may be indicated by absence of a fall in circulating eosinophiles on the ACTH or epinephrine tests, which correlates inversely with adrenocorticoid function. Cortisone, DOCA, salt or other agents are required before and after operation, according to the circumstances.

Chronic adrenal insufficiency in its full-blown form (Addison's disease) is characterized by severe weakness, weight loss, nausea and vomiting, pigmentation, hypotension, and salt and water depletion. Patients with Addison's disease are unusually sensitive to minor stresses and respond poorly to anesthesia and operation. Subclinical forms of the disease are no doubt more common than generally appreciated. Operations should not be undertaken in the presence of overt adrenal failure until metabolic deficits have been corrected.

PREPARATION OF THE PATIENT FOR ELECTIVE OPERATION

It is the surgeon's concern always, in the care of patients, to reduce the calculated risk of surgery to a minimum. A carefully recorded history and detailed physical examination, essential laboratory tests (see Table 13), x-ray studies, and the necessary preparation should precede the *operation of election*. Advantage should be taken of all available knowledge and technics which can be applied to the general proposition of "making the patient safe for the operation, and the operation safe for the patient."

In the final analysis, success or failure of treatment may be determined well before the patient is ever taken from his bed to the operating room. It is not surprising, then, that the scalpel and hemostat, the operative procedure itself, may not be the critical factor in survival.

Recent advances in surgery and new concepts in therapy are based on physiologic data obtained from experimental laboratories, clinical investigation and increasing surgical experience. It is significant that contributions from all of the basic sciences, the anesthesiologist (see Table 14) and the experimental surgeon have been responsible for extending the parameters of *modern surgery* and reducing morbidity and mortality rates to the lowest figures in surgical history.

Adjuncts and safeguards which combine to make operation safer for the patient can be summarized in part as follows:

1. Improved methods for evaluating the operative risk and the newer knowledge of metabolic requirements and body reactions to

and can be treated under local or regional block anesthesia, it is desirable to defer immediate operation. If this is contraindicated (e.g., internal injuries), the stomach must be emptied by intubation (large tube) before the anesthetic is administered. Sedatives, such as chloral hydrate or paraldehyde, are required.

Patients with chronic alcoholism who have taken alcohol in large amounts for prolonged periods often suffer from malnutrition, avitaminosis and other systemic disturbances. Portal cirrhosis (alcoholic cirrhosis) with hepatic insufficiency may develop. The risk of surgery is increased under these circumstances.

Acute alcoholic mania (delirium tremens) sometimes develops after an operation on an alcoholic patient. In about twenty-four hours the patient may become irritable, boisterous, demanding, restless and maniacal. A stage of delirium follows, and finally he recovers or may pass into a coma and die.

Small amounts of alcohol and, if possible, moderate activity may prevent acute mania. Sedatives are helpful but should not be given in excess. ACTH and cortisone have been recommended. Parenteral vitamin B complex is said to be helpful.

Pregnancy

Normal, uncomplicated pregnancy does not in itself increase the risk of operation; nor does the operation itself increase the likelihood of abortion. But there are understandable risks; and the fact that two lives, instead of one, are at stake often makes elective surgical treatment inadvisable during pregnancy. When pregnancy is complicated, the hazards of operation are increased in proportion to the severity of existing metabolic disturbances (e.g., eclampsia gravidarum). Abdominal operations in the later stages of pregnancy are usually difficult technically and should be avoided except under emergency conditions. It should be kept in mind, however, that acute appendicitis develops as frequently among pregnant women as among nonpregnant persons.

Adrenal Insufficiency

Adrenal insufficiency seriously impairs the normal metabolic response to trauma and operation. If unrecognized, the outcome may be fatal; if recognized and treated, the outcome may be favorable. Acute adrenal insufficiency can develop from overwhelming infections with

injury in surgical patients have focused attention on the recognition and correction of preoperative deficits.

2. The fundamental relationship of patient nutrition to patient well-being has been recognized and emphasized. Oral hyperproteinization programs and the use of enzymatic hydrolysates of protein for parenteral use have been introduced to correct existing protein

TABLE 14.—ANESTHESIA CLASSIFICATION OF PHYSICAL STATUS *

I. GOOD: No organic disease—no systemic disturbance.
Examples: Uncomplicated hernias, fractures, etc.

II. FAIR: Moderate systemic disturbance.
Examples: Mild cardiac (I and II), mild diabetes, etc.

III. POOR: Severe systemic disturbance.
Examples: Poorly controlled diabetic, pulmonary complications, moderate cardiac (III), etc.

IV. SERIOUS. Systemic disease threatening life
Examples: Severe renal disease, severe cardiac disease (IV), decompensation, etc.

V. EMERGENCY, GOOD: Patients in Groups I and II with a complication, needing immediate treatment.
Examples. Hemorrhage, open chest wound, perforated viscus, severe respiratory embarrassment, etc.

VI EMERGENCY, POOR: Patients in Groups III and IV with a complication needing immediate surgery.
Examples: Hemorrhage, open chest wound, perforated viscus, severe respiratory embarrassment, etc.

VII SPECIAL: Moribund patients.

* American Society of Anesthesiology, Inc., *Codes for the Collection and Tabulation of Data Relating to Anesthesia, Inhalation Therapy and Therapeutic and Diagnostic Blocks* (1945).

deficits before operation and to maintain nutrition during the period of postoperative starvation.

3. The continued study of body fluid physiology and the clinical investigation of disturbances in water and electrolyte balance have led to better control of parenteral fluid therapy. The practical value of the balance concept has been recognized, the quantitative aspects of volume-for-volume and milliequivalent-for-milliequivalent replacement have received particular emphasis. The significance of dislocations of intracellular and extracellular electrolytes has been recognized More physiologic "repair solutions" are now available for the treatment of mixed water and salt depletion states, acidosis, alkalosis and hypokalemia.

4. Adequate blood-volume replacement before operation, during the time of blood loss and in the postoperative period, with better and safer anesthesia conducted by anesthesiologists, allow the surgeon

TABLE 13.—VALUES FROM ROUTINE DETERMINATIONS IN THE CLINICAL BIOCHEMISTRY LABORATORY *

CONSTITUENT	SAMPLE	NORMAL RANGE
Albumin	Serum	3.8–5.2%
Amylase	Serum	50–150 units
Bilirubin, Van den Bergh	Serum	1 min : 0–0.2 mg. % 30 min.: 0.2–1.0 mg %
Calcium	Serum	9–11 mg. % (4.5–5.5 mEq /L.)
Calcium	Urine	200 mg /24 hr.
Chloride	Serum	600–650 mg. % as NaCl 102–111 mEq /L. as Cl^-
Chloride	Spinal fluid	700–750 mg. % as NaCl 120–128 mEq /L. as Cl^-
Cholesterol, total	Plasma	150–205 mg. %
Cholesterol, esters	Plasma	60–80% of total cholesterol
CO_2 capacity	Plasma	55–70 cc./100 cc plasma 25–32 mEq./L. as HCO_3^-
Creatinine	Blood	1.0–2.0 mg. %
Fibrinogen	Plasma	0.2–0.4%
Globulin	Serum	1.8–3.2%
Glucose	Blood	80–100 mg %
Glucose	Spinal fluid	60–70 mg %
Phosphatase, acid	Serum	0–0.5 units (Bodansky)
Phosphatase, alkaline	Serum	1.5–4.0 units (Bodansky)
Phosphorus	Serum	3.0–5.0 mg % (adults) 4–7 mg. % (children)
Phosphorus	Urine	0.5–1.0 Gm /24 hr.
Potassium	Serum	16–22 mg. %; 4.1–5.6 mEq /L.
Potassium	Urine	1–3 Gm./24 hr.
Protein, total	Plasma	6–8%
Protein, total	Spinal fluid	15–45 mg %
Sodium	Urine	3–5 Gm./24 hr.
Urea nitrogen	Blood	8–18 mg. %
Uric acid	Blood	2–4 mg %
Urobilinogen	Urine	0.5–2.5 mg /24 hr.
Vitamin C	Plasma	0.7–2.5 mg. %
LIVER FUNCTION TESTS		
Bilirubin, Van den Bergh	Serum	1 min 0–0.2 mg. % 30 min.: 0.2–1.0 mg %
Bromsulfalein (BSP)	Plasma	0–10% retention in 30 min.
Cephalin flocculation	Serum	24 hr.. 0–1 plus 48 hr . 0–1 plus
Thymol turbidity	Serum	1–4 units
Urobilinogen	Urine	0.5–2.5 mg./24 hr.
Urobilinogen	Stool	50–250 mg /100 Gm
Zinc sulfate flocculation	Serum	2–8 units
STOOL ANALYSIS		
Amylase	Stool	4 plus activity
Fat, total	Stool	25% of total solids
Protease	Stool	4 plus activity
Total solids	Stool	25% of wet weight
Urobilinogen	Stool	50–250 mg./100 Gm

* Courtesy J. I. Routh, Director, Clinical Biochemistry, University Hospitals, State University of Iowa.

injury in surgical patients have focused attention on the recognition and correction of preoperative deficits.

2. The fundamental relationship of patient nutrition to patient well-being has been recognized and emphasized. Oral hyperproteinization programs and the use of enzymatic hydrolysates of protein for parenteral use have been introduced to correct existing protein

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Examples. Uncomplicated hernias, fractures, etc.

II. FAIR: Moderate systemic disturbance.
Examples: Mild cardiac (I and II), mild diabetes, etc.

III. POOR: Severe systemic disturbance.
Examples. Poorly controlled diabetic, pulmonary complications, moderate cardiac (III), etc.

IV. SERIOUS: Systemic disease threatening life.
Examples: Severe renal disease, severe cardiac disease (IV), decompensation, etc.

V. EMERGENCY, GOOD. Patients in Groups I and II with a complication, needing immediate treatment.
Examples. Hemorrhage, open chest wound, perforated viscus, severe respiratory embarrassment, etc.

VI. EMERGENCY, POOR: Patients in Groups III and IV with a complication needing immediate surgery.
Examples: Hemorrhage, open chest wound, perforated viscus, severe respiratory embarrassment, etc.

VII. SPECIAL: Moribund patients.

* American Society of Anesthesiology, Inc., *Codes for the Collection and Tabulation of Data Relating to Anesthesia, Inhalation Therapy and Therapeutic and Diagnostic Blocks* (1945)

deficits before operation and to maintain nutrition during the period of postoperative starvation.

3. The continued study of body fluid physiology and the clinical investigation of disturbances in water and electrolyte balance have led to better control of parenteral fluid therapy. The practical value of the balance concept has been recognized, the quantitative aspects of volume-for-volume and milliequivalent-for-milliequivalent replacement have received particular emphasis. The significance of dislocations of intracellular and extracellular electrolytes has been recognized. More physiologic "repair solutions" are now available for the treatment of mixed water and salt depletion states, acidosis, alkalosis and hypokalemia.

4. Adequate blood-volume replacement before operation, during the time of blood loss and in the postoperative period, with better and safer anesthesia conducted by anesthesiologists, allow the surgeon

to do long and complicated operations in a safe and unhurried manner.

5. Antibiotic chemotherapy has produced a spectacular decline in morbidity and mortality from the so-called "surgical infections."

6. The value of intermittent or continuous suction-decompression of the gastrointestinal tract has been established. The use of various types of mercury-weighted long tubes has increased the rapidity and success of intubation.

7. Preoperative ambulation the morning of operation, "voluntary cough" exercises, postural drainage, aerosol-antibiotic treatment and systemic chemotherapy, and early ambulation after operation are measures which have lowered the incidence and the seriousness of postoperative pulmonary complications. Tracheobronchial catheter suction and aspiration bronchoscopy are invaluable procedures in the active treatment of postoperative atelectasis.

8. Early ambulation after operation exerts a favorable influence on cardiovascular, respiratory, gastrointestinal, urinary tract and central nervous system functions of the postoperative patient.

9. Anticoagulant therapy and superficial femoral vein or venacaval ligation are contributions to the management of thromboembolic disease, which usually results from "quiet" venous thrombosis in the deep veins of the lower extremities.

10. A postoperative recovery room situated in close proximity to the surgeries makes for economical and efficient deployment of special nursing personnel and the equipment that is required for resuscitation and the early after-care of surgical patients.

Preoperative Orders

The evening before a major operation, written orders are charted which cover the following points:

1. Regarding skin preparation, the orders should specify the extent of the site to be shaved and washed thoroughly with a detergent solution.
2. A saline enema may be given to cleanse the lower bowel if indications exist. This should not be a routine. Mechanical cleansing of the colon with enemas until clear is indicated before anorectal and colon operations.
3. Oral intake after midnight is not allowed if the patient is to have a general anesthetic.
4. The patient should be catheterized before he is taken to the

operating room if there is any question as to complete voiding. It is especially important that the bladder be empty when pelvic or large-bowel operations are planned.

5. Preoperative sedation and medications are ordered. The average effective adult dosages which may be ordered are: pentobarbital, 100 mg., the evening before operation; pentobarbital, 100 mg. orally, or sodium phenobarbital, 100 mg. (H), 60–90 minutes before operation; morphine sulfate, 10 mg. (H) and scopolamine hydrobromide 0.4 mg. (or atropine sulfate 0.4 mg.) 90 minutes before operation.

PREOPERATIVE CARE IN EMERGENCY CASES

Preoperative preparation must often be limited to the bare essentials. Even so, it is never justifiable to omit the details of a carefully recorded history and physical examination. There is usually time for carrying out the most basic and necessary of laboratory examinations and x-ray studies. A few hours spent in preparation may decrease the postoperative morbidity and lower the surgical mortality rate. On the other hand, long delays prove costly in this group of cases. Mistakes in diagnosis can be fatal. In evaluating the *therapeutic perspective* it should be recognized that the *proper timing of operation* may be more of a problem in clinical judgment than is the decision as to the necessity for the operation, the choice of procedure or the details of its performance. The highest premium is paid for technical skill in this group of cases.

The following generalizations can be made about the preoperative treatment of emergency cases.

RESUSCITATION

The treatment of shock by whole-blood replacement, plasma expanders or water-electrolyte solutions is indicated and has priority over all other procedures except the control of exsanguinating hemorrhage, the relief of asphyxial symptoms or the reversal of acute gastric dilatation. Parenteral fluid therapy may be needed as a preliminary to an operation for the correction of dehydration, starvation ketosis with acetonuria, electrolyte depletion, and uncorrected acidosis or alkalosis, as well as oligemic shock. Surgical procedures carried out under gas-oxygen-ether anesthesia are relatively unsafe if the circu-

lating red cell mass is reduced to a point where tissue oxygenation is marginal. Functional embarrassment referable to insufficiency of oxygen is, as a rule, not evident until the hemoglobin has been decreased by approximately 50 per cent. Less than 6 Gm./100 ml. of hemoglobin contraindicates any but the most urgent of operations.

Emptying the Stomach

Tracheobronchial aspiration of blood and vomitus at the time of anesthesia and operation is a serious risk. The endangered group of patients who must be protected from this catastrophe include comatose patients and those with upper gastrointestinal hemorrhage or bowel obstruction. The stomach must be emptied *in every case* before the administration of an inhalation anesthetic for emergency operations. If there has not been a known period of eight to twelve hours of starvation before operation, vomiting should be induced as the most dependable way to empty the stomach. Once vomiting has occurred, a well-lubricated Levin tube is passed into the stomach through one of the nares and the tube is then secured to the cheek with adhesive tape. Intermittent or continuous suction-decompression is then started to keep the stomach empty of fluid and air. The large Ewald tube is sometimes needed to empty the stomach completely.

Decompression of the gastrointestinal tract before operation facilitates the operative procedure, minimizes trauma from the manipulation of a distended stomach or dilated bowel and simplifies the abdominal closure

If there is any question as to the effectiveness of the preoperative measures used to empty the stomach (and clotted blood is particularly difficult to remove), the danger of vomiting and aspiration during the induction phase of anesthesia is best avoided by endotracheal intubation under topical anesthesia.

It cannot be overemphasized that one of the commonest avoidable accidents on surgical services is tracheobronchial aspiration of gastric contents which often proves fatal.

Catheterization

This procedure is indicated before all major abdominal operations involving the pelvic viscera or colon which are carried out as elective operations, and always in the emergency case. Reflex depressor effects from the manipulation of a distended urinary bladder are thus avoided,

and abdominal exploration and the performance of indicated surgical procedures are made easier and less traumatic.

POSTOPERATIVE CARE

The postoperative period may be complicated by frequent and serious difficulties. Fortunately, the introduction of new aids to therapy and a better understanding of the metabolic problems in surgical patients have made this phase less hazardous, but it is no less important than formerly. At the present time, it is possible to anticipate most of the difficulties of the postoperative period and to institute measures which will minimize their seriousness. This is no easy task. It requires attention to all details of treatment, constant watchfulness over the clinical course of the patient and a willingness to start therapy before well-developed signs of trouble exist.

Clinical signposts that are followed from day to day are recorded in the *progress notes* of the patient, and these often give the clue to trouble. These all-important data are:

1. The patient's complaints
2. The general appearance and the clinical progress of the patient —the *directional trend*
3. Trends in body temperature, pulse rate and respirations
4. The nutritional status of the patient
5. The intake-output record of water-electrolyte balance

Procedures at the End of Operation

Clearance is given by the anesthesiologist after aspiration of the airway and a final evaluation as to the *steady state* of the patient. Slow, gentle movement of the patient is essential as he is transferred from the operating table to the stretcher, and later to the hospital bed. The patient is accompanied to the recovery unit by the anesthesiologist, one of the surgical team and a surgical nurse. The postoperative orders are written. Only then can the responsibility for patient care be transferred to the special nurse, who remains in constant attendance until the patient is awake and responding.

Immediate Postoperative Care

Unless a contraindication exists, the unconscious or semicomatose patient should be returned from the operating room to a recovery

ward in a warmed bed. The *dorsal recumbent* or the *lateral decubitus* position with the head lowered offers maximal protection against cerebral ischemia, aids evacuation of the tracheobronchial secretions and protects against the aspiration of blood and vomitus. It aids venous return to the heart and increases the linear blood flow in the deep veins of the legs, a factor which may be helpful in decreasing the incidence of spontaneous venous thrombosis.

During the first six to eight hours after operation, sudden changes in position are not well tolerated by the postoperative patient. This is particularly true under two sets of conditions: (1) if there is a critical reduction in effective circulating blood volume and (2) if regional spinal anesthesia has been used, which obtunds the normal compensatory cardiovascular reflexes against gravity which adjust for the redistribution of blood volume occurring with postural changes. The head-down position may be maintained after operation until the pulse rate and the blood pressure are well stabilized and the patient is considered to be in a steady state.*

The head-down position is not used after extensive operations about the head and neck, or following intracranial operation, because increased venous pressure from the gravitational effect may cause excessive bleeding in the operative wound. It is not used in thoracotomy cases, and obviously, it should not be used if the patient is in respiratory distress, cyanosed or in pulmonary edema.

Except for the first hours after operation (as outlined above), the *head up* and *back up* position is indicated during the course of parenteral infusions, so as to reduce pressure phenomena in the pulmonary circuit which favor pulmonary transudation. This is especially important *in the geriatric patient with cardiovascular-renal disease.*

In general, however, the best position for the sick patient is the one in which he is most comfortable

* Cardiovascular reactions to changes in posture can be related quantitatively to blood loss. This is the basis for the *tilt test* Cardioacceleration and decreased mean arterial blood pressure are proportional to the extent of blood loss. In the normovolemic state, a change from supine to the upright position is associated with a lowered cardiac output in normal individuals of approximately 25 per cent due to decreased venous return The safety factor is reduced and the sensitivity of the test is increased in the patient who has sustained blood loss Clinical evidence of shock in the supine position indicates a probable blood volume deficit of some 20 ml /kg or more and the necessity for a 2,000 ml. (or more) transfusion. An increase in the heart rate of less than 25 beats/minute on tilting indicates a compensated or small blood loss. Increase in heart rate of more than 30 beats/minute suggests a transfusion requirement of approximately 1 L., and syncope suggests the advisability of transfusion in excess of this.

Water and Electrolytes

It is necessary to restrict oral intake of fluid and food for the *first twelve to twenty-four hours after operation*—without exception—if general anesthesia has been used or an abdominal operation has been carried out. By the second or third postoperative day, unless there is cause for persistent ileus, the patient will take fluids by mouth. This amount can be increased, as tolerated, on a progressive schedule, such as:

> Nothing by mouth → 30 ml. warm water each hour → 100 ml. water each hour → clear liquids → full liquids ad lib. → soft diet → general diet.

The foods which may be used are described in Chapter 5, on Nutritional Balance.

Fluid and electrolyte balance are maintained by parenteral therapy during the period of low oral intake. The problems of water and electrolyte balance in surgical patients and the details of parenteral therapy are discussed in Chapter 4, on Fluid and Electrolyte Balance.

Except for the period of complete alimentary starvation which follows immediately after anesthesia and operation, the *feeding schedule* of the patient should be individualized as much as possible. Simple, uncomplicated parenteral therapy of short duration is desirable, and the *very important point of patient safety, as well as the practical* point of economy, is served by an early return to oral intake. Clinical signs of gastrointestinal function which indicate that oral intake can be started with little risk of nausea, vomiting or persistent ileus are: (1) active bowel sounds, (2) absence of abdominal distention, (3) passage of gas by rectum and (4) appetite.

Leg and Lung Exercises

Postoperative atelectasis, pneumonitis and thromboembolism are often preventable complications.

Active leg movements aid venous return and minimize the tendency for quiet venous thrombosis in the veins of the lower extremities. It can be shown that the so-called "bicycle exercises" with active dorsal and plantar flexion of the feet increase linear blood flow in the veins of the lower extremities. Such exercises should be carried out by the patient many, many times during the early days after operation.

Special elastic stockings or elastic bandages which extend to the knee are also helpful.

Frequent turning of the patient from side to side, minimal sedation during the early postoperative phase of postanesthetic depression, supervised deep breathing exercises at frequent intervals during the day and night, a voluntary *forced cough* routine to aid in the removal of secretions in the tracheobronchial tree and early ambulation after operation favor improved aeration and mechanical removal of retained secretions. If patient co-operation is poor and the cough is "wet," tracheobronchial toilet is carried out immediately, using a nasal endotracheal catheter and suction.

Oxygen Therapy

The most common postanesthetic complication is respiratory depression. For this reason, all patients anesthetized with Pentothal,® elderly patients, cardiac cases of the cyanotic group, and many others should have oxygen therapy continuously after the operation until they have reacted completely.

Postanesthetic opiates should be withheld, especially in the older patients, until the patient has recovered from the anesthesia.

When tidal volume falls, eventually the entire reduction in the amount of air breathed is at the expense of alveolar ventilation, since the respiratory dead space remains constant. No significant threat to adequate gas exchange exists until vital capacity falls to 1,000 or 1,200 cc. Respiratory decompensation with hypoxia and CO_2 retention (hypercapnia) develop shortly after this point.

Hypoxia

Since cyanosis may not be present until arterial oxygen saturations fall to 80 per cent or less, the oxygen content of arterial blood (gas analysis or oximeter) provides the only reliable index of low-grade hypoxia. From a practical standpoint, then, the clinician must "think-hypoxia" whenever ventilation or circulation is impaired.

Seldom will the postoperative patient tolerate the *BLB mask* or *Barach metered mask* for the administration of oxygen. The use of an *oxygen tent,* if it is in good service and well managed, is satisfactory; but usually *nasal tube oxygen* is most adaptable. Nasal tube oxygen humidified by bubbling through water is given at a rate of 4–6 L/

minute flow, and alveolar oxygen concentrations of 25–30 per cent are thus realized. Gastric dilatation from swallowed air must be avoided by careful placement of the tube. The catheter is inserted into the nasopharynx until it can be visualized at the uvula, then withdrawn ½ in. and anchored to the face with adhesive tape. Mask oxygen therapy and intermittent positive-pressure respiration (IPPR) is required in the treatment of pulmonary edema.

Hypercarbia (Carbon Dioxide Retention)

As alveolar ventilation falls, oxygen tension falls and CO_2 tension rises in the alveoli and in arterial blood. Since the diffusion constant of CO_2 is not identical with that of oxygen, and because of the greater molecular weight of CO_2, it diffuses less rapidly than oxygen. Although oxygen therapy raises alveolar oxygen tension and arterial oxygen saturation to normal levels by replacing inert nitrogen in inspired air, CO_2 can be eliminated only by increasing the tidal volume or by an excessive increase in the respiratory rate. A shift in blood pH to more acid levels occurs simultaneously with CO_2 retention and an alteration of the $H_2CO_3/BH\ CO_3$ ratio. As a relatively slow adjustment, renal conservation of base and excretion of acid occurs, with an increased CO_2-combining power of blood and acid pH of the urine.

In CO_2 retention there may be anxiety, restlessness, muscular twitchings and rigidity and convulsions. Early in its development the blood pressure and pulse are elevated, but later they fall. The treatment of CO_2 retention is to improve the ventilation by removing causes of hypo-aeration and/or augmenting tidal exchange.

The practical significance of differentiating hypoxia and CO_2 retention from hypo-aeration is obvious.

Postoperative Sedation

The control of pain after operation is accomplished at first with one of the narcotics, *morphine* or *Demerol.®* The shattering of patient morale and the rapid clinical deterioration that results from inadequate pain control indicate the importance of continued attention to this problem. Adequate sedation is needed; yet the excessive use of narcotics and sedatives must be avoided. This is particularly true in geriatric cases ("old age is its own analgesia"), in cases of asthma, hypothy-

roidism, liver disease and in the very young. Thus the problem becomes a highly individualized one which should usually be managed by a single physician. The following generalizations are justified:

1. Morphine or Demerol® is required for the control of postoperative pain in most adult patients during the first day or two after operation Codeine or aspirin should be substituted as soon as possible for the more powerful and habit-forming narcotics.

2. Orders for narcotics must be carefully written, to include (*a*) time limit and (*b*) fixed checks against oversedation. For example, a postoperative morphine order might be written as follows:

> Morphine sulfate, 10 mg. every 4 hours as needed for pain, unless respirations become less than 12/minute; narcotic order limited to 24 hours.

3. Restlessness and sleeplessness, almost physiologic states in the aged, are best controlled with barbiturates, paraldehyde or chloral hydrate. The restlessness of upper-airway obstruction, CO_2 retention, urine retention, intracranial bleeding, alkalosis, uremia, alcohol withdrawal or barbiturate mania do not call for control by the use of sedatives without establishing the cause of the difficulty.

4. The narcotic addict may be hard to handle, but the postoperative period is a dangerous time to submit him to withdrawal therapy. Maintenance doses of narcotics must be given to such patients

Other measures to consider in the control of severe postoperative pain include the following:

1. Intravenous procaine solution must be used in proper dosage and dilution. Dangerous reactions may occur with this drug.

2. Segmental intercostal nerve block, using 1 per cent procaine solution, is easily carried out and affords striking relief of wound pain in almost every instance.

3 Intravenous alcohol solution, 5 per cent alcohol added to dextrose in distilled water has analgesic and sedative effects.

Gastrointestinal Functions

Postanesthetic nausea and vomiting, gas pains and persistent ileus can best be controlled by careful physiologic bowel preparation and the avoidance of drastic purgation preoperatively, early ambulation after operation, improved anesthesia and controlled premedication Early postanesthetic nausea and vomiting may be relieved by the

symptomatic use of such pharmacologic preparations as Benadryl® or Dramamine.®

A rectal tube or rectal irrigation may give effective relief to the patient with gas pains, intestinal distention and anal sphincter spasm.

The diagnosis of *fecal impaction* is suggested by pain in the rectum or a sense of rectal fulness, painful bowel movements, constipation or the incontinence of a diarrhea-like stool. Digital removal of the impaction is required, together with repeated mineral oil retention enemas. Fecal impaction is a special problem in the patient who has had barium x-ray studies of the gastrointestinal tract without complete evacuation of the barium mixture before operation; the patient with peptic ulcer who is receiving aluminum hydroxide or Sippy powder management; and in those cases where a lower abdominal operation, perineal or anorectal procedure has been done.

Suction-decompression of the gastrointestinal tract, intermittent or continuous, using a *nasogastric tube* or a mercury-weighted *long tube* (Miller-Abbott, Kaslow, Cantor or Harris), is indicated after a variety of operations. These include cases of elective resection for small- or large-bowel obstruction, biliary tract surgery, gastric resection, peritonitis, acute abdominal injuries and many other conditions requiring surgery. The outstanding complications of suction-decompression are water and electrolyte problems. Disturbances in fluid-electrolyte balance can be avoided in such cases only if an accurate volume-for-volume and milliequivalent-for-milliequivalent replacement of losses is made.

Acute Urinary Retention

Because of the increased risk of infection, the urinary bladder should not be allowed to become overdistended.

Catheterization of the postoperative patient is indicated in the following situations:

1. Given a chance to void first, and unable to do so, debilitated and elderly patients should have placed indwelling a self-retaining Foley type catheter as a precautionary measure against urinary retention during the early postoperative period.

2. If there is inability to void with evidence of bladder distention and lower abdominal distress, the patient should be catheterized.

3. The incontinent patient should be catheterized so as to minimize the risk of tissue maceration and bedsores from the combination of

wetting and pressure. Catheter management simplifies the nursing problem in such cases.

4. After abdominoperineal resection, extensive perineal plastic operations and the repair of vesicovaginorectal fistulas, catheterization should be done.

5. An inlying catheter is indicated if hourly *serial urine volume determinations* are desired as a measure of cardiovascular-renal hemodynamics (acute dehydration states with oliguria, oligemic shock, the burned patient).

6. After injury (traumatic or surgical) to urinary bladder or urethra, in combination with other procedures, the patient should be catheterized.

Early Ambulation after Surgery

The favorable influence exerted on a number of body functions by early ambulation is evidenced by decreased muscle wasting, improved muscle tone, shortened convalescence and improved patient morale. From a practical viewpoint, the nursing load per patient is reduced by early ambulation after operation.

The principle of early ambulation can be followed in the majority of surgical cases. There is no experimental or clinical evidence to show that it has an unfavorable effect on wound healing. The contraindications to ambulation are (*a*) peritonitis, (*b*) serious cardiac disease with congestive heart failure or myocardial-coronary insufficiency, (*c*) insecure wound closure and (*d*) thromboembolic disease.

A *standardized regimen* of early ambulation has been described by Leithauser (1951). *It is presented, with some modifications,* as follows:

1. The patient should be ambulatory before the operation, as well as in the postoperative period.

2. As soon as he recovers from anesthesia, ambulation may be started. At least four walk-periods are required on the day after operation. At first, the patient walks only a short distance; this is then repeated until the limit of tolerance is reached. The technic of mobilizing the patient for the first time is as follows. The patient is moved into the lateral position at the edge of the bed, then he flexes the thighs and knees so that the lower extremities are brought over the edge of the bed. He then is helped to a sitting position by rotation through a lateral plane. This maneuver results in least tension on the wound and a minimum of wound pain. A change to the erect position will often be followed by paroxysmal coughing The patient is encouraged to breathe deeply and to cough out tracheobronchial secretions. There may be

transient light-headedness and vertigo. Steps are placed at the bedside to avoid sharp strain on the wound as the patient stands; he should wear shoes or a slipper with a heel, so as to avoid foot and leg strain. He walks about for a few minutes; then the return to bed is accomplished in reverse order. The patient should be assisted in performing these maneuvers the first two or three times.

3. During rest periods in bed, leg and lung exercises are performed every half-hour. In the first stages after operation, the patient is not allowed to sit in a chair for prolonged periods. Sitting may lead to venous stasis and encourage thrombosis. It should be emphasized that the patient must walk as much as he feels he can and then return to bed.

4 The patient walks to the bathroom as soon as he is able. Postoperative enemas are seldom required, and use of the bedpan is uncommon.

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CHAPTER 11

Care of the Patient in the Operating Room

ONCE THE decision to operate has been made, certain arrangements are necessary. The operating-room staff must be notified, the anesthesiologist contacted and, in consultation with him, the anesthetic agent and technic to be used determined, the premedication ordered, and the preliminary preparation of the operative field carried out before the patient is transported to the operating room.

CHOICE OF THE ANESTHETIC AGENT AND TECHNIC

The choice of the anesthetic agent and technic must be based on a knowledge of the properties of the drugs used and the advantages and disadvantages of the various technics. The selection should be made taking into consideration all factors which pertain to the individual patient.

There are three fundamental factors to be considered: (1) the safety of the patient, (2) the convenience of the surgeon and (3) the comfort of the patient These are listed in the order of their importance; and if sacrifices are necessary, they must be made in the second and third factors. Only rarely must the safety of the patient be sacrificed for the convenience of the surgeon or for the patient's comfort

In regard to the safety of the patient, not only should the time during which the patient is anesthetized be considered but also the effect of the agent or technic on the postoperative period. The choice of the agent and technic should therefore be on a long-range basis and not confined to the anesthetic period alone. For example, for an

obese patient who is to have a cholecystectomy it might be better to use a quick-acting agent, such as cyclopropane, in preference to ether because the postanesthetic somnolence and debility accompanying the slow elimination of ether might predispose the patient to pulmonary complications.

The requirements of the surgeon can generally be met if these are made known. For this reason, the selection of the agent and technic should be a joint decision by the surgeon and the anesthesiologist. Such items as the type of operation, the relaxation required, the length of the operation, the presence or absence of cautery or other electrical equipment (explosion hazard) and personal preferences will influence the choice. The anesthesiologist is obligated to satisfy the requirements of the surgeon within the limits of safety to the patient. It is essential, therefore, that there be close co-operation between the surgeon and the anesthesiologist.

The patient is primarily interested in his own comfort. His impression of good anesthesia is based on his own or another's experiences and relates to pleasant, rapid induction and a recovery period devoid of distress. While it is often possible to promise these small measures of comfort, it is also unwise to accede to these demands when they would prove unsafe. Comfort for the patient should be the factor which is most readily sacrificed in favor of convenience to the surgeon and, especially, safety to the patient.

The following questions are helpful in making a selection:

1. What is the safest agent and technic for this patient for this operation?
2. What are the surgical requirements?
3. How can the patient be made most comfortable?

PREANESTHETIC MEDICATION

Depressant drugs are widely used to provide analgesia, sedation or diminution of autonomic nervous system activity. Their administration must be based on a reasonably precise evaluation of the needs of the patient and a working knowledge of the pharmacologic capabilities of the drugs. The desired effects may be obtained with one drug or, if necessary, a combination of two or more drugs. Specific indications should be established for each drug in the combination. A "shotgun" type of therapy is poor practice.

The drugs commonly used are representatives of three groups:

the opiates and their substitutes, the barbiturates and the belladonna derivatives.

Morphine reduces oxygen consumption (lowers metabolic activity), elevates the pain threshhold and produces a diminution in the response to noxious stimuli. It should be used in premedication and for all general purposes only when these properties are desired.

Morphine may be administered by any of three routes, depending on the time available for the production of its full effect. If given subcutaneously, the full effect is secured in approximately ninety minutes; if given intramuscularly, in about forty-five minutes; and if given intravenously, in about fifteen minutes.

Substitutes for morphine include: Pantopon,® meperidine (Demerol,® Pethidine), methadone, Dilaudid® and Levo-Dromoran.® Substitutes have been sought in an effort to eliminate or diminish respiratory depression, addiction, parasympathomimetic action and other side reactions of morphine. For the most part, there has been little success in these attempts, and morphine continues to be widely used

Barbiturates are employed when there is need for sedation (sleep or hypnosis), protection against the convulsive effects of cocaine and similar drugs, and as an anticonvulsant. These drugs are poor agents to use for pain relief. When given in the presence of pain, the usual inhibitions controlling responses to pain are so altered that a severe state of disorientation often results.

Drugs of the belladonna group are employed to decrease the output of secretions from the upper respiratory tract, depress or eliminate reflexes mediated through the vagus, or produce some sedation or amnesia Atropine and scopolamine are the drugs commonly used. Scopolamine has certain advantages over atropine: it is more effective in counterbalancing the respiratory depression of morphine; it contributes a satisfying measure of psychic sedation and amnesia; and it inhibits the nausea associated with morphine.

The route of administration of the belladonna group drugs is governed by the desired time of onset of action. When given subcutaneously, the onset is within a half hour; when given intravenously, the onset is almost immediate. The drugs act for approximately three to four hours. As with morphine, it is desirable to have the full effect developed before the induction of anesthesia As a rule, atropine or scopolamine are given at the same time as morphine. They may be combined in the same syringe.

The dosage of depressant drugs required is influenced by a number of factors. These include: the potency of the drug, the physical state of the patient and his level of metabolic activity, the type of anesthetic to be used, the amount of drug previously given the patient, the amount and type of other drugs to be used, etc. Routine practices should be avoided, and the choice of drugs and doses must be adjusted for each individual patient.

Ultimate determination of the dosage depends on the individual's metabolic activity, which varies with age. The most important single factor influencing the estimation of the level of reflex irritability, and therefore the tolerance to depressant drugs, is the patient's age. Metabolic activity increases sharply from birth to five years of age, then decreases slightly and rises again at puberty, after which it gradually declines until old age. The reason for frequent overdosage in children and the aged must, therefore, be apparent.

The basic levels of metabolism are increased by fever, pain, emotional disturbances and specific hypermetabolic states (hyperthyroidism). Patients with such complications have a higher tolerance for depressant drugs and require larger doses than do other patients. The basic levels of metabolism are decreased in certain races (Negroes, Orientals) and by debilitating diseases and hypometabolic states. Under these circumstances, tolerance to depressant drugs is decreased and smaller doses required

The premedication required for a healthy adult might consist of 10 mg. morphine sulfate and 0.4 mg. scopolamine hydrobromide (or atropine sulfate 0.4 mg.), subcutaneously ninety minutes before operation; intramuscularly forty-five minutes before operation; or, if the intravenous route is chosen, about two-thirds of the above dose well diluted in saline fifteen minutes before operation.

If spinal, nerve block, infiltration or topical anesthesia is to be used, a barbiturate should be administered to minimize the *toxic* effects of *local anesthetic drugs* and to provide psychic sedation. Pentobarbital sodium may be used. The usual dosage is 100 mg. orally sixty to ninety minutes before the operation.

All the foregoing dosages are based on the amount of drug required to produce the desired effects in a healthy adult male. The amount prescribed in any given individual must always be based on a consideration of the factors which influence the response to drugs; these factors have been only briefly described in this section.

PARENTERAL THERAPY

An intravenous setup should be available to permit the infusion of a variety of solutions (dextrose, 5 per cent, in distilled water, isotonic saline, Pentothal,® vasopressor substances, muscle relaxants, plasma, plasma-volume expanders, or whole blood) as needed. A three-way stopcock arrangement should be introduced into the setup, or other provision made for the administration of parenteral medications. The setup must be arranged so that rapid forced transfusion of blood can be accomplished at any time, without difficulty or delay. If the intravenous needle is insecure, the saphenous vein at the ankle may be exposed and cannulated, preferably with a polyethylene tube of suitable caliber.

PROTECTION OF THE PATIENT

Although the surgeon's responsibility for the patient's welfare is shared in the operating room with the anesthesiologist and the rest of the surgical team, it can never be transferred completely to others. The surgeon must make sure that each member of the team is on the alert to protect the patient It should be emphasized that, as a result of anesthesia, the patient is in a critically vulnerable state. He must be protected against the special risks in the operating room, such as the possible adverse effects of the room's temperature and humidity on the heat-regulatory mechanism and the possibility of injuries resulting from the posture during the operation

Because the body's heat-regulatory mechanism is impaired by anesthesia, it is necessary to guard the patient against the loss and retention of body heat. Operating-room temperatures should be maintained at about 75° F. If the temperature and humidity are high or the operation is prolonged, water and electrolyte losses by vaporization and sweating may be large.

The *supine horizontal position* is the position best tolerated by the anesthetized patient. In normal breathing, the thoracic cage enlarges in all directions except posteriorly. With the deeper planes of anesthesia, the intercostal part of the respiration tends to be abolished and most of the load of breathing is shifted onto the diaphragm. Changes in ventilation because of the effects of surgical position result from: (*a*) diminished excursion of the diaphragm during the expiratory phase as a result of loss of abdominal muscle tone; (*b*)

factors such as undue retraction on the wound, abdominal packs, tight binders, the flat prone or steep Trendelenburg position, which cause elevation of the diaphragm by increased intra-abdominal pressure; and (*c*) reflex effects of position, such as might result from the patient resting on his lower ribs in either the prone or lateral position. Circulatory distress results from, first, venous and capillary stasis in dependent extremities. Reduced venous return results in lowered stroke-volume and cardiac output, loss of support of vein walls due to paresis and hypotonia of muscles of the abdominal wall and extremities, reduced suction and force-pump action on the great veins caused by reduced respiratory movements, and loss of vasomotor tone under anesthesia. So long as the effects of gravity are annulled and the patient is quiet, he is all right. But changes from the horizontal position are dangerous.

The therapeutic corollaries from such considerations are obvious. The following practices should be observed: Avoid sudden changes in position. Use small pillows beneath the pelvis and shoulders to minimize interference with respiratory exchange. Avoid the deeper planes of anesthesia. Confine the use of braces or the kidney rest to selected cases and do not allow pressure against the ribs on the dependent side. Employ supplemental breathing if ventilatory exchange is inadequate.

The *surgical prone posture* hinders respiratory movement somewhat. When an anesthetized patient is placed lying with the face downward on the firm, flat surface of an operating table, diaphragmatic movements are impeded by the weight of the body resting on the abdomen. In the prone posture, freedom of diaphragmatic movement is favored by slight elevation of the pelvis on a pillow placed transversely under the anterosuperior iliac spines. Endotracheal intubation is advisable for patients who are in the prone position or in any other position which hinders respiratory movement or makes control of the airway difficult.

The *jacknife position* with arms, legs and head dependent tends to reduce venous return to the heart. Costal excursion is reduced by pressure on the ventral surface of the thorax; and compression of the abdomen with increased intra-abdominal pressure interferes with respiratory exchange and the pumping action of the diaphragm, causing decreased venous return.

The *lithotomy position* with extreme flexion of the thighs plus the Trendelenburg position is poorly tolerated by obese patients, owing

to respiratory embarrassment resulting from increased intra-abdominal pressure. Moreover, when the legs are lowered, redistribution of the blood volume into the extremities and visceral regional circulations may lead to severe hypotension.

The *Trendelenburg position*, if it is steep, reduces cardiac output. There is interference with diaphragmatic movement, as well as changes in intraperitoneal pressures which make ventilation more difficult

The *lateral kidney position*, by pressure on the thorax, interferes with intercostal breathing on the dependent side. Venous return from all the extremities is retarded by gravity. Reflex effects may occur.

The high *gallbladder* rest used with the *cholecystectomy position* requires profound anesthesia in order to relax the stretched abdominal muscles. Interference with venous return from the kidneys and lower extremities, owing to compression of the inferior vena cava against the hyperextended vertebral column, may occur. Relaxation of back muscles and forced hyperextension may be responsible for postoperative back pain.

The *sitting position* interferes the least with costal respiration. An abdominal binder is recommended for patients with relaxed abdominal muscles, since such muscles contribute to diaphragmatic inhibition. However, deep anesthesia should not be used with the patient sitting, because blood will stagnate in the extremities.

Neurovascular injuries may follow malposition on the operating table. The principal factor in the development of such injuries is ischemia, resulting from stretching or pressure on nerves or vessels. Remember that the anesthetized patient is pain-free and cannot complain about postural insults. Muscle tone is reduced by anesthesia, and so the patient is more susceptible to the effects of unnatural positions. Trauma to nerves or vascular structures can occur under these conditions, and the damage is usually not discovered until the postoperative period.

The brachial plexus is especially vulnerable if the steep Trendelenburg position is used with no shoulder braces or with poor placement of the shoulder braces This is particularly so if the arm is hyperextended on a board for the operation, as is done for radical mastectomy cases, for the administration of parenteral fluids or simply in making room for the surgeon's assistant. If the shoulder brace is allowed to press against the soft structures in the supraclavicular space, rather than over the bony acromioclavicular joint, injury may result.

The radial, ulnar and median nerves are vulnerable to trauma because they traverse the brachium. If the arm hangs over the edge of the operating table, the nerves may be compressed between the humerus and the edge of the table. The same injuries may occur if the surgeon's assistant leans against the arm, crushing it between his body and the operating table.

Foot-drop from common peroneal nerve paresis and paresthesias due to pressure on the saphenous nerve can be attributed to the lithotomy position and pressure of the metal braces which hold the stirrups, or pressure in the popliteal fossa from the leg braces on obstetrical tables.

Peripheral vascular damage may also occur. Subclavian or axillary artery compression and occlusion may result from long-sustained pressure in hyperabduction. Venous thrombosis in superficial veins may be the result of braces, padding or straps which cause venous occlusion.

CARE OF RESPIRATION AND CIRCULATION

No anesthesia is safe or satisfactory unless diligent efforts are made to maintain a functioning airway. There are no short cuts for avoiding constant attention to the patency of the airway.

A free airway is one through which the inspired atmosphere passes, without obstruction, from outside the body into the arterial blood and through which waste gases can pass, without obstruction, from the venous blood to the external atmosphere. The airway includes the naso- and oropharynx, the larynx, the trachea, the bronchi and all subdivisions, and the alveoli.

An obstruction to the airway produces several undesirable effects: it calls for increased effort on the part of the patient, it makes it difficult to obtain and maintain anesthesia; and it seriously interferes with oxygen intake and CO_2 elimination. While the patient may tolerate the increased respiratory effort occasioned by an imperfect airway and may not suffer too greatly from an uneven and inadequate anesthesia, he will die if the airway obstruction is great enough or prolonged enough to impede his oxygen intake and CO_2 elimination.

Methods of Improving and Maintaining the Airway

Suction.—No anesthesia (including all technics) should be induced, maintained or terminated without having a source of suction

available. The need for removal of foreign material from the pharynx, larynx or tracheobronchial tree must always be anticipated, and ready dependable suction is the most efficacious method of accomplishing this. Various aspirating appliances, such as a metal tip, a catheter with a "whistle tip," and an ordinary urethral catheter, may be used.

SUPPORT OF THE JAW AND POSITION OF THE HEAD.—Interruption of the air flow in the oropharynx can be prevented or corrected without mechanical means by preventing the mandible from falling back and by not allowing the tongue to sag. This can be done by grasping the mandible (at its midpoint) between the thumb and forefinger and pushing it forward (Fig. 49). Traction on the tongue with the fingers, suture or forceps will also relieve the obstruction but is traumatic and no more effective.

The head of the relaxed patient is in the optimal position when it is turned to the side and slightly extended (Fig. 50). This maneuver often leads to improvement in the airway. Turning the patient into the lateral position also will often provide notable improvement in the airway and facilitate drainage of mucus, blood, etc., from the pharynx. A slight head-down position is likewise helpful in establishing and maintaining a patent airway. Do not put the patient's head in a position with the face up, and do not use a pillow unless it is placed well under the shoulders and the neck is not flexed.

MECHANICAL AIRWAYS.—Two devices commonly used to correct obstruction to air flow in the upper air passages are: the oropharyngeal airway (several types) and the nasopharyngeal airway. The manner of inserting the oropharyngeal airway is shown in Figure 51.

CLEARING THE AIRWAY AT THE LARYNX

The best treatment for laryngospasm is prophylactic treatment. Adduction of the vocal cords is a protective mechanism to prevent contamination of the lower respiratory tract. Laryngospasm can always be traced to errors of omission or commission in the administration of the anesthesia. Sudden high concentrations of anesthetic agents should be avoided, agents which would enhance the laryngeal reflex should not be used in situations in which the laryngeal reflex is apt to be activated, and foreign material should be kept out of the oropharynx. If there is likelihood of contamination of the oropharynx, or of laryngospasm, it is best to prevent adduction of the cords by introducing a tracheal catheter.

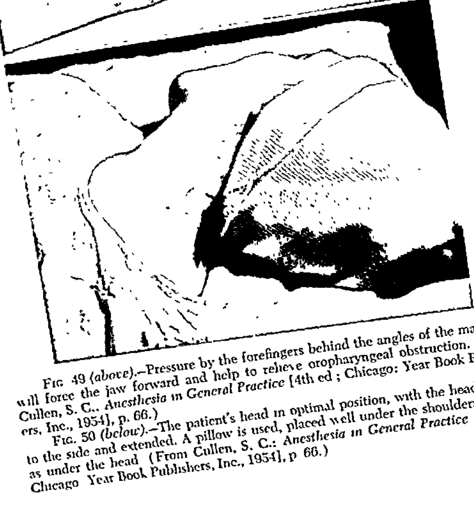

FIG. 49 (*above*).—Pressure by the forefingers behind the angles of the mandible will force the jaw forward and help to relieve oropharyngeal obstruction. (From Cullen, S. C., *Anesthesia in General Practice* [4th ed ; Chicago: Year Book Publishers, Inc., 1954], p. 66.)

FIG. 50 (*below*).—The patient's head in optimal position, with the head turned to the side and extended. A pillow is used, placed well under the shoulders as well as under the head (From Cullen, S. C.: *Anesthesia in General Practice* [4th ed ; Chicago Year Book Publishers, Inc., 1954], p 66.)

Laryngospasm, once present, must be treated promptly and effectively. The cause or causes of the spasm must be determined and immediate steps taken to reverse the situation. The concentration of the anesthetic agent must be reduced by dilution with oxygen; manipulations which may be inciting the spasm must be removed; and if the reflex is being hyperactivated by an intravenously administered barbiturate, the drug should be discontinued.

If the oropharyngeal airway is patent, it is often possible to force

FIG 51.—Method of inserting an oropharyngeal airway. The forefinger of the left hand is placed between the buccal surface and the alveolar ridge behind the last tooth and gently rotated The airway is inserted with a rotating motion over the tongue. (From Cullen, S C: *Anesthesia in General Practice* [4th ed.; Chicago Year Book Publishers, Inc, 1954], p. 69.)

oxygen through the spastic cords by means of a mask fitted snugly to the face and intermittent pressure applied by hand to a bag full of oxygen After a little oxygen is forced past the adducted cords, the relief of anoxia causes relaxation of the spasm and the patient resumes *voluntary breathing. The bag and mask should be left in place* and oxygen supplied until there is certainty that the anoxia has been completely relieved.

This method of control of laryngospasm is usually sufficient. *Tracheal intubation should be rigorously avoided at this time.* The time required to make such an attempt will add to the hypoxia and may be the critical factor leading to mortality. Unless there is a mechanical obstruction, tracheotomy is likewise inadvisable. Manual

methods of artificial respiration are ineffective in the presence of severe laryngospasm and should not be used until the airway is clear.

Clearing the Airway in the Trachea and Bronchi

Relief of a blockade of the airway at this point usually entails the removal of foreign material, such as blood, pus, mucus, vomitus and foreign bodies. This is best accomplished by bronchoscopy, but in emergencies the insertion of a tracheal catheter through which a suction catheter can be directed is quite effective and often lifesaving.

The patient should be placed in the head-down position with the head turned to one side. Sometimes, pounding the patient on the back will assist in the expulsion of foreign bodies. Infants and small children can be held by the feet in a completely inverted position for short periods to facilitate drainage of the tracheobronchial tree.

Ventilation

The establishment and maintenance of adequate ventilation is also of fundamental importance. The anesthesiologist must make an accurate estimate of normal ventilation for the individual patient, must be alert to the effects of hypoventilation and be familiar with the factors predisposing to it and must take steps to correct deficiencies in ventilation when they occur.

The anesthesiologist is intimately concerned with ventilation because the patient's welfare requires that respiratory exchange take place. In inhalation anesthesia, it is necessary to provide for movement of the anesthetic gases and vapors to and from the point at which exchange takes place between the alveolar atmosphere and the blood.

Treatment of Hypoventilation

Ventilation can be accomplished simply and effectively by manual intermittent positive pressure applied to the rebreathing bag. It is important that the pressure be so applied that the mean pressure throughout the respiratory cycle is minimally elevated. There is evidence that elevation of the mean pressure interferes with the return of venous blood to the heart and causes a reduction in cardiac output. This condition is usually observed only in patients with a deficient circulatory status.

There are other mechanical devices which are used to assist ventilation. Some of these produce intermittent positive pressure, and others produce intermittent positive and negative pressure. They effectively aid ventilation but have the disadvantage of being less adaptable to minute-to-minute changes in the requirements of the patient.

GENERAL ANESTHESIA

Inhalation Anesthetic Agents

The drugs used to produce anesthesia by inhalation may be volatile or gaseous. *The volatile agents are liquid at atmospheric pressure* but volatilize to the vapor state on exposure to the atmosphere; they are administered as a vapor. The gaseous agents are in liquid form at high pressure but change to the gaseous state on exposure to the atmosphere; they are administered as a gas.

The volatile anesthetic agents commonly used are: ethyl ether, divinyl ether (Vinethene®), chloroform, ethyl chloride and trichloroethylene. The gaseous agents are: nitrous oxide, ethylene and cyclopropane.

To facilitate selection for particular anesthetic problems, these drugs can be further classified according to their physical and pharmacologic properties. The physical properties which are of interest are: the type, the weight of the vapor or gas in relation to the air, the flammability, the odor and the stability. The pharmacologic properties of practical interest are: the potency, duration of action, the safety ratio, and the effect on various organs and the autonomic nervous system.

It is beyond the scope of this work to delve into anesthetic pharmacology, but it must be emphasized that a working knowledge of the basic physical and pharmacologic properties of these agents is essential to their usage. The student is urged to consult works on this subject.

Inhalation Anesthetic Technics

A rational appreciation of inhalation anesthesia is obtained if one remembers that the exchange of all gases and vapors is dependent on the gradients in partial pressures of these gases and vapors Each succeeding medium through which the gas passes will attempt to attain the partial pressure of the gas or vapor in the preceding medium The rate at which this equilibrium in tension is secured depends on the

ability of the gas or vapor to traverse biologic membranes. This ability depends chiefly on the density of the gas or vapor, but the passage of the gas or vapor is also influenced by the state of the membrane. The rate at which equilibrium is established depends also on the extent to which gases are moved in the lung and the extent to which exchange is influenced by mechanical mixing or diffusion. Ventilation of the lung is an essential factor.

The level of anesthesia depends on the blood level of the anesthetic drug. Depression of nerve tissue is in direct proportion to the blood level of the agent, and the level of the agent is in direct proportion to the partial pressure of the agent to which the plasma is exposed as it passes through the lung.

The maintenance of anesthesia by the inhalation of gases or vapors is influenced by the solubility of the particular agent in lipoid tissues. The more potent agents (ether, Vinethene,® cyclopropane) are highly soluble in fat; therefore it is necessary to add the agent to the blood to replace that lost from the plasma to the lipoid tissues. An equilibrium between the plasma and lipoid tissue levels is reached as the anesthesia progresses, so that less and less is lost from the plasma to the fat tissues; and therefore, less and less agent need be added to the inspired atmosphere to maintain a satisfactory blood level.

Recovery from inhalation anesthesia depends on the same factors that influenced induction and maintenance, only in reverse order. When the mask is removed, the tension of the anesthetic agent in the inspired atmosphere drops to zero, and the flow of gas or vapor is then from the tissues to plasma, to alveoli to air, assuming that there is competent ventilation and circulation. The rate at which desaturation occurs depends on the solubility of the gas or vapor in the blood and its partial pressure in the tissues.

Several inhalation systems and technics are available for the administration of anesthetic gases and vapors. These range from the simple "open drop" technic to the "closed" technic. In the former, the drug in liquid form is vaporized on gauze or a face mask; and the drug, mixed with air, is inhaled. In the closed system, the gas or vapor is confined to a closed system to which oxygen is added as required and from which CO_2 is removed by chemical absorption with "soda lime." Rebreathing of the anesthetic agent occurs. Variations between these two technics are called "semiopen" or "semiclosed," according to whether a reservoir bag is used or rebreathing occurs. In all methods, there must be a source of oxygen, a means for disposal

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and hypnotic properties rather than their anesthetic properties, since they are not analgesics. They are properly utilized in combination with nitrous oxide or spinal, regional or local analgesia. Under some conditions the muscle relaxants are used in conjunction with the short-acting barbiturates.

The most widely used agent is Pentothal® sodium. Because of the ease of administration and the relative lack of disagreeable side effects, this agent has become popular with patients and physicians alike. However, it is a dangerous drug and, once given, cannot be reclaimed. It influences the vagus nerve to effect an increase in the laryngeal reflex, to constrict the bronchioles and dilate the spleen. If high blood levels are attained, it is prone to produce a fall in blood pressure. An increase in the laryngeal reflex with resultant laryngospasm constitutes its greatest hazard and contraindication.

Pentothal® should not be used in any patient in whom patency of the airway cannot be assured at all times. Unless intubation is used (the tube may be introduced under topical anesthesia before the barbiturate is given), Pentothal® should be avoided if there is likelihood of incitement of laryngospasm by pus, blood, mucus, vomitus or other foreign material in the respiratory tract. It should not be administered to patients who must be placed in the prone position. It should be used with extreme caution in patients subjected to procedures likely to incite reflex laryngospasm (e.g., dilatation of the anal sphincter and stripping of the periosteum). Generally, Pentothal® is contraindicated for patients in shock or in whom shock may be anticipated.

Operations on the head and neck, chest and abdomen, especially if relaxation is required, usually contraindicate the use of the short-acting barbiturates as the chief anesthetic agent. Their use should be confined to short procedures in patients in good condition where relaxation is not a prerequisite and where the likelihood of adverse reflexes and impairment of the airway is remote. Even under ideal conditions, apnea and laryngeal obstruction may develop. The physician must therefore always be prepared to cope with these emergencies.

REGIONAL ANESTHESIA

Spinal Analgesia (Anesthesia)

Spinal analgesia is that state of analgesia secured by the introduction intrathecally of procaine or a similar drug. The analgesia is

of CO_2 and a device for administering the anesthetic gas or vapor.

The selection of the technic depends on a number of measurable factors, the proper application of which greatly influences the success and safety of the anesthetic procedure. Among these factors are: the heat loss, humidity of the inspired air, CO_2 retention and resistance to ventilation.

Muscle Relaxants

There are several drugs (e.g., Flaxedil,® d-tubocurarine, succinylcholine, decamethonium) which, by virtue of their ability to produce muscular relaxation, enhance the safety of anesthesia and the convenience of the surgeon. They are not anesthetic drugs, but they are effective by blocking neuromuscular transmission.

A satisfactory anesthetic state should be established before attempting to augment relaxation by the administration of relaxants. Many of the difficulties associated with the use of these agents are related to insufficient anesthesia. Excessive and protracted respiratory paralysis and bronchospasm often result from attempts to provide relaxation under inadequate anesthesia The muscle relaxants are adjuncts to well-conducted anesthesia and are not the means for overcoming deficiencies of poorly managed anesthesia.

All of the relaxants are given intravenously. They may be used in conjunction with any of the inhalation agents, keeping in mind the additive effect of d-tubocurarine and ether. The dose required varies with the extent of relaxation needed, the degree of relaxation provided by the anesthetic agent and the type of relaxant used.

Neostigmine and physostigmine are effective antagonists to curare and Flaxedil ® Their ability to antagonize curare appears to depend largely on a strong anticholinesterase property. Tensilon® also has an anticurare effect through its competition with curare at the cholinergic receptors in the neuromuscular junction.

The complications associated with the use of the muscle relaxants involve both the respiratory and the circulatory systems Depression or paralysis of the respiratory system is most common.

Intravenous Anesthesia

The ultra-short-acting barbiturates given intravenously (Pentothal® sodium and Evipal® sodium) are widely used for their depressant

tension. There are means of preventing the hypotension in most patients, and there are effective means of treating it once it has developed; but one cannot guarantee that hypotension will not appear, despite adequate prophylactic measures. For this reason, it is not advisable to use spinal analgesia for patients in impending or overt shock

The constant significant incidence (5–10 per cent) of postlumbar puncture headache is a complicating factor which seriously interferes with the pleasant postanesthetic course of spinal analgesia. There is also a small, but real, incidence of neurologic complications, which are often minor and short-lived but which may be major and protracted.

Regional, Infiltration and Topical Analgesia

Regional nerve block is accomplished by interrupting the nerves with procaine or similar drugs at a site distant from the site of the proposed operation. *Infiltration analgesia* is accomplished by blocking the nerves at the site of the proposed operation. *Topical analgesia* is obtained by blocking the nerve endings by the application of an agent on the surface.

These types of analgesia are useful when it seems advisable to anesthetize small areas with minimal interference of general physiologic mechanisms. They are also useful when it is advisable to reduce to a minimum the chances of postanesthetic nausea and emesis—for example, in plastic reconstructions about the face and neck, ophthalmologic surgery and fixation of mandibular fractures. With these types of analgesia it is possible, and sometimes beneficial, to retain the co-operation of the patient. Some patients prefer regional, infiltration or topical analgesia to technics which produce unconsciousness. Complete relaxation can be secured by the proper use of these technics, and bleeding during the surgery is probably minimized.

There are also certain disadvantages to these technics A patient must be co-operative and able to distinguish between sensations of touch, pressure, traction and pain. Some patients are incapable of differentiating these sensations and are not good subjects for regional or infiltration analgesia. As a general rule, any patient who does not want analgesia by either of these methods is a poor candidate.

Proper premedication is a prerequisite to satisfactory regional, infiltration and topical analgesia and many times makes the difference between good and poor results. The use of morphine and scopolamine intravenously is extremely beneficial during analgesia produced by

confined to the area supplied by the nerve roots affected by the drug, and it may be associated with muscular relaxation and paralysis of the fibers of the sympathetic nervous system which happen to traverse the roots involved in the anesthesia. Although only a portion of the patient is patently anesthetized, spinal analgesia must be considered, in a sense, a general anesthesia, in that changes induced by the paralysis of the nerve roots indirectly influence the whole body. This influence on the body as a whole is directly proportional to the number of segments that are anesthetized; i.e., the higher the level of spinal analgesia, the more profound the general effect.

It is generally believed that the interruption of the transmission of nerve impulses is accomplished in the nerve root after it leaves the spinal cord and before it exits from the dura, and that anesthetization of the tracts of the spinal cord is not affected. The degree to which the nerve roots are anesthetized is, in a broad sense, entirely dependent on the concentration of the drug to which they are exposed. The different modalities of sensation and the fibers controlling muscular and sympathetic tone are paralyzed in a relatively constant order. Pain sensation disappears early; touch, kinesthetic sense and deep pressure sense disappear later, and in about that order. Block of the sympathetic fibers takes place before the appearance of analgesia, and muscular relaxation develops shortly afterward. It is entirely possible, and sometimes advantageous, with low concentrations of the drug, to produce good analgesia and sympathetic paralysis without significant degrees of muscular paralysis.

Spinal analgesia has several features which make it a desirable technic It is capable of effecting extreme muscular relaxation. It causes contraction of the small intestine by interruption of the sympathetic pathways. This contraction facilitates exposure within the abdomen Spinal analgesia is useful when it is expedient to employ cautery or electrical apparatus during surgery. It is also useful when the patient's co-operation is desired during the operative procedure and in patients who do not like to lose consciousness. The postanesthetic period is somewhat less distressing than that following other technics, because of a decreased incidence of nausea and emesis.

There are also certain disadvantages to spinal anesthesia. Except in continuous spinal analgesia, the drug cannot be retrieved once it has been introduced into the subarachnoid space, and any complications that may develop must be treated symptomatically. There is always the likelihood of precipitous and inordinate degrees of hypo-

tension. There are means of preventing the hypotension in most patients, and there are effective means of treating it once it has developed; but one cannot guarantee that hypotension will not appear, despite adequate prophylactic measures. For this reason, it is not advisable to use spinal analgesia for patients in impending or overt shock.

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Proper premedication is a prerequisite to satisfactory regional, infiltration and topical analgesia and many times makes the difference between good and poor results. The use of morphine and scopolamine intravenously is extremely beneficial during analgesia produced by

these methods. Morphine enhances the analgesia and helps to minimize the discomforts of position, pressure and traction. Scopolamine is helpful in dissipating some of the apprehension that is present in some patients. On occasions, it is useful to produce light hypnosis with Pentothal® The induction of analgesia is often time consuming, and the length of action of the drugs is limited.

Regional, infiltration or topical analgesia should be chosen on the basis of the three fundamental principles which are helpful in selecting other technics. In making the choice, it is well to remember that these technics are often inconvenient to the surgeon, frequently are discomfiting to the patient and add little to the patient's safety. Their use is confined most practically to those situations in which the co-operation of the patient is needed, the establishment and maintenance of the airway by artificial means is difficult and unwieldy, the postoperative nausea and emesis endanger the patient, the proposed surgery is minor or the patient is moribund. These technics should not be forced on patients except in the rare instances in which the safety of the patient would otherwise be imperiled They have a valuable and useful function in anesthesiology, but there is a general tendency to overrate their safety features.

There are certain fundamental requirements for consistently successful regional, infiltration and topical analgesia which cannot be ignored. The anesthesiologist must have a complete working knowledge of the surgical anatomy of the area he intends to block. He must be familiar with the properties of the agents used and be trained in the recognition and treatment of untoward reactions to these drugs. He must have a properly prepared and co-operative patient, and the surgeon must be aware of the necessity for gentleness in the surgical manipulations.

REACTIONS TO LOCAL ANESTHETICS

There seems to be a popular, but erroneous, impression that reactions to procaine and similar drugs are due to a sensitivity of the patient and that the reactions are allergic in nature. It is safe to say that 99 per cent of these reactions are *not* anaphylactic or allergic in nature. If they are, the reaction is characterized by the sudden onset of wheals, edema, hypotension and other phenomena associated with this type of reaction from any cause. If a patient gives a history which includes symptoms and signs of this order, it is advisable to avoid the use of

the offending drug or of one with a similar chemical structure. But if a patient gives a history of, or is observed in, a reaction of the types outlined later, it is not necessary to avoid repetition of the drug. The incidence of true allergic reactions to procaine or similar drugs is extremely low.

The usual reaction to procaine or similar drugs is due to the absorption of a sufficient amount of the drug into the blood stream to cause symptoms and signs of toxicity in direct proportion to the blood level, and the rapidity of its attainment. This is a fundamental principle which, if kept in mind, will clarify the prophylaxis, recognition and treatment of such reactions.

All of these anesthetic drugs are toxic, and their toxicity is increased in a geometric ratio as the concentration of the drug increases. In other words, a 2 per cent solution of procaine is not just twice as toxic as a 1 per cent solution, but about four times as toxic. Also, absorption of these drugs is increased in areas in which the circulation is abundant. Examples of such areas are the mouth and pharynx, the urethra and the caudal canal. A good general rule for prophylaxis is to avoid, whenever possible, the use of high concentrations of these drugs in areas where absorption into the blood stream will be rapid.

An immediate reaction is due to accidental intravenous injection of the analgesic solution or to absorption of a large quantity in a very short time. The reaction appears within a few seconds after the injection or topical application of the drug, and it is characterized by abrupt and complete deterioration of circulation and respiration. The patient may be dead within a minute or two.

Most reactions appear after five to fifteen minutes. If a reaction has not developed within thirty minutes, it is not likely to develop at all. The symptoms and signs appear with varying degrees of rapidity and severity, depending on the rate of absorption and the quantity absorbed.

Some patients become uncomfortable and apprehensive. Others become euphoric and elated. This latter reaction occurs most frequently after the application of cocaine. The physician should talk to the patient during the induction of regional, infiltration or topical analgesia in order that premonitory signs of a reaction may be detected.

When drowsiness or other signs develop early, one should check the pulse. It may be of good quality and approximately the same rate as it was before the injection, or it may be of poor quality and somewhat slower than normal. If the pulse is poor in quality, the blood pressure should be checked. It will be lower than normal if the reaction is

progressing. Hypotension may become extreme. Associated with the hypotension will be pallor, perspiration, clammy skin and syncope Respiratory irregularities, twitching about the face and in the fingers, and soon a full-blown generalized convulsion may appear. Respiratory and circulatory failure follows the convulsions if they are not controlled.

The foregoing symptoms and signs comprise the complete reaction Minor degrees of this major reaction may occur, depending on the blood level of the drug.

Patients in whom only the mental changes develop require no *therapy. The injection or topical application of the drug* should, of course, be discontinued. One should, however, be alert to the progression of symptoms and signs and be ready to institute treatment if the reaction becomes more severe.

The hypotension must be treated with a vasopressor drug. The drug is given preferably intravenously to insure a prompt and effective response. The vasopressor drug should be administered in repeated doses (every three to five minutes) until satisfactory blood pressure is obtained. In severe states of hypotension, massive total doses of vasopressor drugs may be needed to restore and stabilize the blood pressure. It is extremely important to elevate the pressure, because persisting low tensions may cause enough tissue hypoxia to produce permanent damage.

Oxygen therapy should be given. This helps to minimize the danger of anoxia during the period of hypotension and respiratory irregularities. A mask and bag are useful, particularly if assistance to pulmonary ventilation is required. Endotracheal catheterization is beneficial in the presence of severe reactions.

Twitchings and convulsions are controlled by the intravenous administration of a soluble barbiturate, such as Pentothal,® which is usually readily available. Enough barbiturate must be given to stop the convulsion; but this amount may stop respirations, in which case artificial respiration can be carried out. It is mandatory that the convulsions be stopped.

Cardiac Arrest; Ventricular Fibrillation

Cardiac arrest and ventricular fibrillation are infrequent but important operating-room emergencies. They occur in healthy, good-risk patients as well as in the debilitated, often when least expected, and are fatal unless promptly recognized and vigorously treated. For these

reasons, it is essential that the surgeon and the anesthesiologist be keenly aware of the possibility of the development of cardiac arrest, the need for avoiding those circumstances which may provoke it, the signs by which it is recognized and the measures for immediately correcting it.

Cardiac arrest may follow the severe bradycardia of asphyxia, or it may result from the reflex effects of trauma to intrathoracic or intra-abdominal structures. Adequate oxygenation and removal of CO_2, as well as the avoidance of sudden increases or high concentrations of anesthetic agents, are important factors in preventing cardiac disturbances under anesthesia. The use of procaine in the mediastinum is of some practical importance in preventing reflex cardiac standstill or ventricular fibrillation due to operative manipulations about the heart and great vessels, the hilar structures and the esophagus.

Fortunately, ventricular fibrillation that requires special electrical defibrillating equipment is relatively uncommon. It results, most typically, from direct injury to the heart or from the reflex effects of chest wall trauma, or it is superimposed on pre-existing heart disease. Certain anesthetic gases and drugs, notably cyclopropane and epinephrine, potentiate the development of cardiac irregularities.

In treatment, *time is of the essence,* and success will be measured in minutes and seconds. Irreversible brain damage results after approximately three minutes if there has been complete arrest of the cerebral circulation. The urgent need in either cardiac emergency—arrest or fibrillation—is *a manually sustained cardiac output* and *controlled respiration* The surgeon and anesthesiologist must be forearmed:

An orderly, sequential procedure for diagnosing and treating cardiac arrest follows:

1. The diagnosis of a cardiac emergency requiring resuscitative measures is based on the clinical evidence of sudden failure of pulse and blood pressure. The absence of pulsation in the major vessels and/or an ashen pallor is sufficient for the working diagnosis of cardiac arrest or ventricular fibrillation, and no delay is allowable.

2. If the chest is not already open, an immediate left transverse transthoracic incision (no special consideration is given for asepsis) to the heart through the 4th or 5th intercostal space should be made. The costal cartilages above and below are divided and the ribs spread apart. There will be no bleeding.

3. The pericardium should be opened at once and cardiac massage i.e., cardiac compression, begun, compressing the heart rhythmically

at about 40–80/minute, which will allow for adequate ventricular filling.

4. Meanwhile the patient should have been placed in the head-down position, in order to obtain the beneficial effect of gravity on cerebral blood flow and venous return.

5. Positive-pressure artificial respiration with 100 per cent oxygen is needed because of the open-chest conditions established by the thoracotomy. Mouth-to-mouth insufflation may be used until suitable equipment is available, should the emergency arise elsewhere than in the operating room.

6. Forced and rapid intravenous infusion of whole blood or plasma is indicated. Intra-arterial transfusion, using a femoral artery, or the aorta if the chest is open, is a more direct but less often used approach to the problem of perfusing the coronary and cerebral circulations. Intermittent compression of the aorta distal to the left carotid is beneficial in improving flow to these vital areas.

7. A 1 per cent procaine solution should be used in the pleural and intrapericardial spaces to minimize reflex effects on the heart and to reduce cardiac irritability fom direct injury due to cardiac compression.

8. A direct-writing electrocardiogram record should be obtained as soon as possible. If ventricular fibrillation exists, defibrillation is essential. Successful resuscitation requires the application of an electric current to arrest all activity before an effective beat can be resumed spontaneously.

9. *Direct injection into the right atrium or into a peripheral vein* of epinephrine solution is useful for *increasing the strength of contraction of a failing heart which has only recently resumed a spontaneous beat.* In the adult, 0.1–0.5 cc. of a 1:1,000 solution in 5 or 10 cc. of isotonic saline is used But use cautiously; it can cause fibrillation.

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CHAPTER 12

Postoperative Complications

THE POSSIBILITIES for difficulty during the postoperative period are numerous. Some of the more common complications, according to their sites of origin, are given in Table 15. In most instances, however, recovery from properly planned and conducted

TABLE 15.—COMMON CAUSES OF POSTOPERATIVE MORBIDITY AND MORTALITY

I. *Pulmonary Complications*
   1. Bronchitis, lobar or lobular atelectasis, massive collapse, pneumonia and pneumonitis
   2. Aspiration pneumonia
II. *Gastrointestinal Complications*
   1. Postanesthetic nausea and vomiting, acute gastric dilatation, gas pains, postoperative ileus
   2. Postoperative obstruction
   3. Fecal impaction
III. *Urinary Complications*
   1. Postoperative retention
   2. Acute renal insufficiency
   3. Urinary tract infection
IV. *Cardiovascular Complications*
   1. Hemorrhage and shock
   2. Cardiac complications
   3. Phlebothrombosis, pulmonary embolism and thrombophlebitis
V. *Wound-Healing Complications*
   1. Hemorrhage
   2. Wound infection
   3. Wound separation, superficial and deep (disruption)
VI. *Miscellaneous*
   1. Postoperative hiccups
   2. Postoperative parotitis
   3. Peritonitis and intraperitoneal abscesses

surgical treatment is relatively uneventful. Much depends on the application of the principles of surgical care described in Chapter 10; but despite the surgeon's best efforts, complications may develop. Often they are, in themselves, of minor consequence; but if allowed to persist or progress, they assume major importance and jeopardize the patient's recovery. It is necessary, therefore, that the student have a clear understanding of the pathogenesis of minor, as well as the major, postoperative complications, the means by which they are recognized and the measures by which they are averted or treated.

PULMONARY COMPLICATIONS

Pathophysiologically, most postoperative pulmonary complications are related. Atelectasis is the most common and most important of these conditions. The other complications in this group include bronchitis, massive collapse, and postoperative pneumonitis and pneumonia. At one time they were referred to as "ether pneumonias," but it has been shown that the anesthetic agent per se is not the responsible cause.

The present concept of the pathogenesis of postoperative pulmonary disorders emphasizes the role of obstructive phenomena. Obstruction to the tracheobronchial tree by mucus secretions ("plugs") leads to the entrapment of air in the distal pulmonary tissue. The gases in the obstructed segment are absorbed through the intact circulation, and alveolar, segmental, lobar or complete pulmonary collapse results. The secretions of the air passages usually harbor bacteria, which then secondarily invade the collapsed area or areas, producing pneumonitis. On the basis of the pathogenesis of atelectasis, the implications for therapy are clear: to minimize those conditions which favor the production and retention of tracheobronchial mucus, to facilitate removal of secretions by the patient and, if obstruction occurs, to evacuate the mucus and re-expand the collapsed areas of lung.

The factors which favor development of atelectasis and other lung complications in the postoperative period are: reduced or impaired ventilation; ineffective coughing or depression of the cough reflex by wound pain, narcotics, etc.; infrequent changes in position during the postoperative period; and pre-existing respiratory infections.

Diaphragmatic movement normally accounts for more than half of the pulmonary ventilation. This is reduced, after abdominal operations, by voluntary and reflex spasm due to wound pain and irritation. Gener-

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diagnosis of postoperative atelectasis is warranted if a sudden unexpected elevation in the temperature, pulse and respiration occurs. The temperature may spike to 104° F. or higher, and there will be a concomitant elevation in the pulse and respiratory rates. The severity of these signs may suggest the magnitude of the pulmonary involvement. Usually there is a productive cough, with expectoration of thick and tenacious sputum which is never blood tinged. The presence of tracheo-

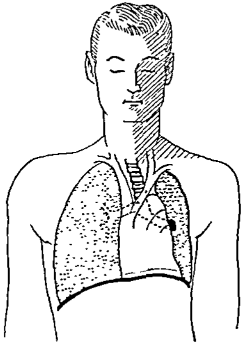

FIG. 52.—Massive pulmonary collapse (atelectasis) producing secondary intrathoracic shifts. Note the mucus plug obstructing the left mainstem bronchus and *the airless left lung. The mediastinum has shifted to the left; the trachea, great vessels* and heart have moved to the left; the left leaf of the diaphragm is elevated and the left hemithorax is retracted. The right hemithorax is relatively overexpanded.

bronchial phlegm is often apparent if one simply listens to the patient's breathing. Dyspnea and cyanosis are prominent when massive collapse has occurred.

The physical signs will depend on the extent of the collapse of the pulmonary tissue. The signs of *extensive collapse* (Fig. 52) are:

1. There is decreased or absent chest expansion on the affected side.

2. There is displacement of the point of maximum impulse of the heart (mediastinum) toward the involved side, unless the atelectatic

ally, the nearer the operative site is to the diaphragm, the greater will be the decrease in ventilation and the higher the incidence of pulmonary complications. The incidence of atelectasis is therefore particularly high after operations on the stomach, biliary tract, pancreas and spleen. It is said that men are predominantly abdominal breathers, while women are thoracic breathers. This may account for the greater frequency of these complications in the male.

The depressant influence of wound pain on cough and depth of respiration can be observed immediately after abdominal and thoracic operations. The patient is usually unable to inspire normal volumes of air and cannot expire forcefully. The commonly used analgesic drugs also decrease the effectiveness of these defenses. The belladonna derivatives tend to dry up secretions and hence increase their viscosity and tenaciousness. These drugs are valuable in the preoperative period but contraindicated in the postoperative period. Tight abdominal dressings or binders and abdominal distention often further impair the effectiveness of coughing and deep breathing.

Pulmonary complications commonly follow operations undertaken in the presence of respiratory infections. The combination of inflammation, increased secretions and pathogenic bacteria provides an ideal setup for the development of atelectasis and pneumonitis. For this reason, it is advisable to defer elective operations on patients suffering from acute or chronic respiratory infections until complete or maximal recovery has been attained. If postponement is impossible, then all measures necessary to protect against the development of pulmonary complications should be used.

Postoperative bronchitis, if unrecognized and untreated, may lead to atelectasis and pneumonitis. It is characterized by excessive mucus in the respiratory passages, a persistent productive cough, a low-grade fever and such physical signs as rhonchi ("rattle") coarse râles and diminished ventilation.

Postoperative pneumonitis (pneumonia) is likely to be superimposed on persistent bronchial obstruction and atelectasis and is produced by bacterial invasion of the collapsed lung. The involvement may be localized or diffuse.

Postoperative Atelectasis

The danger period is the first forty-eight hours after operation, during which time the clinician must "think atelectasis." A presumptive

general supportive measures should be instituted. The technic of endotracheal aspiration is described in Chapter 31, on Chest Injuries.

GASTROINTESTINAL COMPLICATIONS

The common and related postoperative conditions which are evidence of disturbed gastrointestinal function are: (1) postanesthetic nausea and vomiting, (2) acute gastric dilatation, (3) gas pains and (4) postoperative inhibition (paralytic) ileus.

Physiologic disturbances of intestinal motility occur regularly after operation and injury and represent a normal reaction to stress. When they persist or become aggravated, they are clinically significant and require treatment. The many local and general factors influencing the development of gastrointestinal dysfunction are not detailed here. Suffice it to say that basically the neuroendocrine control mechanisms mediate these responses. (See Chapter 3, on The Systemic Response to Injury.)

Gastrointestinal dysfunction is most likely to be associated with purgation of the bowel before operation, oversedation and prolonged anesthesia, trauma to the bowel; peritoneal irritation from any cause; shock, dehydration and electrolyte imbalance; and malnutrition with hypoproteinemia and hypovitaminosis.

Because severe functional obstructions tend to be self-perpetuating and progressive, the need for prevention and early treatment should be apparent. This in turn depends on constant watchfulness during the postoperative period, periodic auscultation of the abdomen, early recourse to the scout film of the abdomen and a willingness to start active treatment early.

If unrelieved, functional or organic intestinal obstructions may result in irreversible changes. The distention vicious cycle is initiated and sustained, reflexly at first, then perpetuated by the effects of mechanical stresses: distention, distention-necrosis, bowel perforation and peritonitis. Free or fixed kinks from intestinal distention may cause closed-loop intestinal obstruction. If there is intraperitoneal inflammation, distention favors the development of serosal agglutination, adhesions and mechanical blockade.

Symptoms and Signs

The warning signs of gastrointestinal complications after operation are: (1) persistent nausea and vomiting, (2) abdominal distention,

process is bilateral or the mediastinum is fixed by previous inflammatory changes.

3. There may be hyperresonance to percussion and diminished to absent breath tones before collapse occurs. Later, with consolidation, there is percussion dulness, tubular or amphoric breathing and râles.

4. The x-ray changes consist of increased density of the collapsed pulmonary tissue, mediastinal shift toward the affected side and elevation of the diaphragm with narrowing of the intercostal spaces on the involved side.

Prevention

Elective operations on patients suffering from acute upper respiratory infections should be deferred until recovery has occurred. Patients with chronic pulmonary disease should be treated with postural drainage, bronchoscopy, antibiotic drugs, locally (aerosol) and/or systemically until maximal improvement has been achieved.

The stomach must be empty before operation, in order to decrease the danger of vomiting and aspiration. Prolonged deep-plane anesthesia and postoperative depression must be avoided. The injudicious use of narcotics favors the development of pulmonary complications. Transverse abdominal incisions appear to cause lesser degrees of ventilatory impairment than vertical incisions. Tight binders and tight dressings applied to the abdomen and chest predispose the lungs to trouble During the immediate postoperative period, frequent changes of position, voluntary or induced (carbon dioxide–oxygen) deep breathing and coughing, and early ambulation are important. The shock position favors drainage of the tracheobronchial mucus. If the mucus is tenacious, steam inhalations and mucolytic agents are helpful. Ammonium chloride 0.6–0.9 Gm every four hours orally, or sodium iodide 1.0 Gm. three *times a day, added to the intravenous solutions, may be used.* Ambulation is most important The upright position aids ventilation and increases the effectiveness of cough.

Treatment of Postoperative Atelectasis

The prophylactic measures described above may be effective in clearing out retained secretions and restoring air to the collapsed air sacs. Supplementary measures include: vigorous fist percussion to the affected area of the chest, endotracheal aspiration, and bronchoscopic aspiration if indicated. In addition, antibiotics, oxygen therapy and

regress. It may be described as a disorganization and impairment of the motor function of the gastrointestinal tract, which sometimes results in complete cessation of motor activity of the bowel and functional obstruction.

The signs are: a silent abdomen (to auscultation); evidence of scattered gas, both in the small and large bowel on the abdominal scout

FIG. 53.—Plain (scout) film of the abdomen in advanced paralytic ileus secondary to peritonitis from a ruptured appendix. Note the excessive distention of many loops of bowel, from gas. On the patient's right side, the cecum, the ascending colon and a portion of the transverse colon are easily made out. In the midabdomen there are distended loops of small intestine. A nasogastric tube is in the stomach.

film (Fig. 53); a notable absence of cramping abdominal pain; distention of the abdomen; and passage of little or no gas or fecal material by rectum

The principal aims of treatment are to sustain or restore normal metabolism and to relieve intestinal distention If either objective is realized, it will favorably influence the other. The measures commonly used for correcting postoperative ileus are: (*a*) suction-decompression of the gastrointestinal tract with the short or long intestinal tube, and nothing by mouth; and (*b*) drug therapy. Prostigmin® (neostigmine),

(3) abdominal pain ("gas" pains), (4) auscultatory evidence (silent abdomen) and (5) x-ray evidence of obstruction.

VOMITING.—Vomiting in the immediate postoperative period is generally of short duration. Continuous nasogastric suction becomes necessary if the vomiting is persistent. If it does not respond quickly to this therapy, other conditions must be considered: vomiting from metabolic imbalances (fluid, salt, acid base, uremia, etc.), vomiting from peritonitis or obstruction and vomiting from central nervous system disturbances (e.g., cerebrovascular accidents).

ACUTE GASTRIC DILATATION.—Acute gastric dilatation may be a part of the reflex inhibition of the gastrointestinal tract. Mechanical occlusion of the third part of the duodenum may result from direct pressure of the dilated stomach or by arterial mesenteric compression where the superior mesenteric artery crosses over the duodenum. The stomach is soon distended with accumulated secretions and swallowed air.

Symptoms usually appear within the first forty-eight hours. There is persistent belching, hiccups and retching. The patient may complain of epigastric fulness.

Vomiting is of the "overflow" type; that is, the stomach is never emptied completely. Characteristically, the patient will vomit small amounts of foul-smelling, greenish or black fluid at frequent intervals. Bile, changed blood and residual food account for the appearance of the vomitus. The danger of aspiration is great. Acute dilatation of the stomach may be responsible for otherwise unexplained shock, and even death.

The abdomen is distended and tympanitic, especially over the epigastrium. A succussion splash may be elicited. The diagnosis is established by gastric intubation and aspiration.

The preventive treatment consists of early mobilization, use of nasogastric suction until there is evidence of return of gastrointestinal function, and avoidance of excessive oral intake. Attention must be given to the correction of fluid and electrolyte deficits.

GAS PAINS.—A lack of co-ordinated activity between the segments of the bowel is a fundamental factor in the production of gas pains. The condition may appear with the resumption of motor activity after the period of physiologic ileus. Gas in the large bowel, which is often responsible for abdominal distress, may sometimes be relieved by the use of a rectal tube or rectal irrigations.

POSTOPERATIVE INHIBITION ILEUS.—This is a poorly defined entity said to exist when milder degrees of gastrointestinal dysfunction fail to

THROMBOEMBOLIC PHENOMENA

The term "thromboembolic phenomena" is applied to conditions characterized by venous thrombosis and secondary involvement of the lung by embolism. Although not necessarily confined to the postoperative period, these conditions occur at this time with great regularity and contribute significantly to the morbidity and mortality of operative treatment. Venous thromboses often develop in the veins of the lower extremities, where they frequently lead to permanent disturbances in the circulation. When a large thrombus becomes an embolus and lodges in the pulmonary artery, it produces serious, and even fatal, alterations in cardiorespiratory function.

Phlebothrombosis ("quiet venous thrombosis") occurs predominantly in the deep veins of the calf muscles, the veins of the plantar muscles of the foot and pelvic veins, where a noninflammatory loosely attached thrombus is produced. The thrombus often propagates centrally into larger femoral and iliac veins. Segments of the clot may become detached and carried free in the blood stream to the lungs.

Thrombophlebitis may occur in any vein, but the veins of the extremities are most often involved. The clotting process is usually initiated by trauma, infection or chemical irritation (e.g., prolonged infusion of a hypertonic solution). A local inflammatory reaction is produced and thrombosis follows. Perivenous inflammation results in vasospasm, edema, pain and tenderness. The clot usually is anchored to the vein wall and shows little tendency to become detached. The local changes in the leg from interference to the circulation are often of more concern to the physician than the danger of embolism. But pulmonary embolism can also occur in thrombophlebitis.

Etiology of Thromboembolic Phenomena

The changes which lead to thrombophlebitis are organic in nature and inflammatory in origin; those which lead to phlebothrombosis are probably functional in character. The changes are incompletely understood, but they comprise all alterations which may initiate clotting. They include:

1. Blood changes after operation: increase in platelets, "stickiness" of platelets, alterations in fibrinogen, increased blood viscosity, leukocytosis, acceleration of the clotting mechanism from dehydration, plasma loss, etc.

0.5 mg. every four hours, has been recommended as a prophylactic measure after operation. In certain situations, if judiciously used, it may be indicated as a therapeutic adjunct, but it probably has limited value.

Other practical measures consist of the use of colon irrigation, sometimes in conjunction with Prostigmin.® The rectal tube alone will often provide a vent through a spastic anal sphincter area and give relief.

Seldom used measures include: splanchnic anesthesia, intravenous

TABLE 16.—Differential Diagnosis of Postoperative Ileus and Postoperative Obstruction

	Postoperative Ileus	Postoperative Obstruction
Surgical history	Usually develops within seventy-two hours of operation	Operation may have preceded onset by months or years
Abdominal pain	Slight or absent	Frequent, cramping, and may be severe
Nausea and vomiting	Present	Present
Abdominal distention	Present	Present
Fever	*Frequent, because the* ileus is often associated with peritonitis	*None or slight,* unless complicated
Auscultation	Silent abdomen	Noisy abdomen: audible borborygmi, peristaltic rushes and high-pitched tinkles
Scout film	Diffuse scattered loops of distended bowel (usually small and large bowel)	Single or multiple localized loops of distended bowel (usually small bowel)

hypertonic saline solution, enterostomy drainage, diffusion-decompression by inhalation of 100 per cent oxygen and the local application of ice bags.

Postoperative Obstruction.—The early differentiation of functional *obstructions from mechanical obstructions is essential, for proper* therapy is dependent upon it. Functional obstructions require nonoperative treatment; whereas established organic obstructions require operative relief. In the postoperative period, edema, fixation, angulation or inflammatory exudate may lead to mechanical obstruction, partial or complete. Often when relief of bowel distention has been accomplished, continuity and function are re-established. The salient points in the differential diagnosis of these conditions are listed in Table 16.

include: the maintenance of blood volume and water and electrolyte balance; the treatment of local foci of infection before operation; avoidance of hypotension; leg exercises after operation; shock position if there is postoperative depression; avoidance of restricting abdominal or thoracic binders; prevention of increased intra-abdominal pressure; the use of supportive bandages to the lower extremities; and

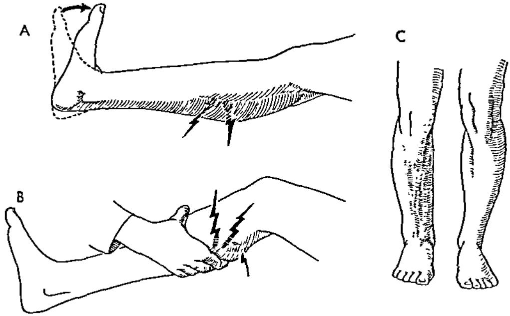

FIG. 54.—Signs of phlebothrombosis of the calf muscle veins. *A*, Homans' sign, pain in the calf on dorsiflexion of the foot with the leg in extension. *B*, tenderness of the calf muscles on compression *C*, slight swelling about the ankle and prominence of the veins.

early ambulation. Prophylactic anticoagulant therapy may be indicated if the patient gives a history of previous thrombosis.

ACTIVE TREATMENT

THROMBOPHLEBITIS.—The patient should be kept at bed rest with the extremities elevated. External heat, usually in the form of warm wet packs will give relief from pain and will hasten resolution of the inflammation. Antibiotic therapy should be instituted if there is reason to believe infection is present. Paravertebral procaine block of the lumbar sympathetic chain relieves pain, releases vasospasm and improves circulation to the part. Anticoagulant drugs are useful in halting the clotting process.

2. Factors which favor circulatory retardation: immobility, hypopnea from wound pain, shock or sustained hypotension, abdominal distention, tight abdominal binders, Fowler's position, etc.
3. Injury to the vein wall. There is reason to believe that certain physicochemical alterations in the intima may lead to spontaneous clot formation. The exact nature of these changes is unknown, but they appear to be concerned with the endothelial cell membrane and/or the intercellular cement substance.

Clinical Picture

Thrombophlebitis.—The clinical manifestations of thrombophlebitis are the result of inflammation and vasospasm. There are fever, tachycardia and other evidences of a systemic reaction. The area of redness, increased heat and tenderness may be confined to a visible superficial vein. Edema and other evidences of circulatory impairment result from mechanical and functional interference with blood and lymphatic flow. Vasospasm may be severe and may involve both arteries and veins. The clotting process may extend into other superficial veins or into deep veins. The involved veins are often cordlike.

Phlebothrombosis.—This is usually a dangerously silent process The signs and symptoms which indicate the presence of phlebothrombosis (Fig. 54) are: pain in the calf muscle; slight edema of the pretibial area, tenderness of the calf muscle on *gentle* compression; pain in the calf on dorsiflexion of the foot with the leg extended (Homans' sign), increased prominence of the superficial veins of the foot and lower leg, and persistent slight elevation in body temperature and pulse, in the absence of other findings, beyond the first three or four days after operation. Often the process is silent. The patient may have no pain in the leg, and there may be no significant physical signs. Under these conditions, phlebothrombosis may be missed unless the physician is acutely aware of its insidiousness and makes repeated careful examinations. Sometimes the diagnosis is made on suspicion alone or after an episode of pulmonary embolism.

Prevention

The prevention of thromboembolic accidents is a therapeutic ideal. This is approached by the institution of a number of precautionary measures, before, during and after operation. These measures

leg (either thrombophlebitis or phlebothrombosis) is the "postphlebitic syndrome." This disabling condition is characterized by chronic swelling, pigmentation, varicosities, eczema and ulceration of the extremity. (See Chapter 28, on Peripheral Vascular Diseases.)

PULMONARY EMBOLISM.—Most pulmonary emboli arise from venous thrombi of the lower extremities (Fig. 55) and pelvis. In more than half the cases there is no apparent reason for release of the clot; in other cases, slight physical exertion (e.g., sitting up in bed, straining on a bedpan, getting out of bed and walking) appears to precipitate embolism. The number and particle size of the clots which reach the pulmonary bed are related directly to the clinical manifestations. A single small embolus may cause no significant signs. A shower of small emboli may mimic heart disease or pleurisy. A large embolus produces asphyxial symptoms with sudden agonizing chest pain, dyspnea, cyanosis, shock and death.

Infarction of the lung follows massive embolism or develops from progressive thrombosis of the pulmonary veins after embolic occlusion. It does not always occur after embolism; therefore, failure to demonstrate an area of pulmonary infarction by x-ray does not rule out previous embolic episodes.

In pulmonary infarction there is cough and hemoptysis, pleuritic type pain, x-ray evidence of a wedge-shaped infarct and sometimes delayed jaundice from red cell breakdown, if the patient survives. Death from pulmonary embolism results from shock, asphyxia, acute cor pulmonale, impaired venous return to the left side of the heart, widespread visceral reflex effects or a combination of these. Not uncommonly, the diagnosis of pulmonary embolism is missed until necropsy.

Treatment of Pulmonary Embolism.—Generally, there is time for treatment. Although the mortality from massive embolism is high, 60 per cent of the fatal cases survive beyond one hour after onset of symptoms. Emergency treatment includes:

1. Absolute bed rest.
2. Oxygen therapy.
3. Anticoagulants. Heparin should be given immediately to prevent propagation of the clot.
4. Drugs:
   (a) Morphine, 0.008–0.016 Gm.
   (b) Atropine, 0.0008–0.001 Gm.
   (c) Papaverine, 0 03 Gm.

These drugs should be given intravenously. Atropine and

PHLEBOTHROMBOSIS.—Bed rest, elevation of the legs, anticoagulant drugs and, occasionally, sympathetic blockade are indicated. Ligation of the superficial femoral vein (or vena cava) should be considered when a warning episode of pulmonary embolism has occurred, or when, in the clinician's judgment, the condition cannot safely be

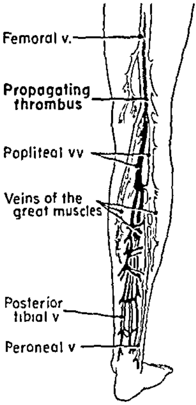

FIG. 55.—The deep veins of the leg are the usual primary site of quiet venous *thrombosis* (phlebothrombosis). Note the unattached noninflammatory clot extending *into the* superficial femoral vein Propagation of the clot into the iliac veins or the vena cava can occur. The danger of pulmonary embolism is great. (After Homans)

controlled by anticoagulant therapy. The exact place of venous ligation and anticoagulant drugs in the treatment of phlebothrombosis has not been clearly established. Neither venous ligation nor anticoagulant therapy will protect completely against pulmonary embolism. Because phlebothrombosis in one leg is often followed by its appearance in the *opposite* leg, simultaneous bilateral superficial femoral vein ligation is usually advisable.

The most serious late complication of venous thrombosis of the

or should spontaneous hemorrhage occur, measures for reversing this tendency must be instituted.

URINARY COMPLICATIONS

Postoperatively, the most frequent urinary tract complications are: acute urinary retention, acute renal failure and infection.

Urinary Retention

Temporary inability to empty the bladder often occurs after abdominal, pelvic, perineal and urinary tract operations. It occurs chiefly in adults and most often in males. If uncomplicated, it is a distressing but not a serious problem.

Many factors contribute to urinary retention. There may be atony of the detrusor muscle and spasm of the external sphincter muscle. There is often decreased sensitivity of the bladder reflex from drugs, anesthesia, manipulation and pre-existing urinary obstruction. Because of the loss of bladder sensitivity to the distention which normally initiates the reflex, spontaneous emptying may be impossible. Stagnation of urine favors the development of infection. Therefore, to prevent infection, overdistention of the bladder and retention of urine must be prevented.

The following factors may contribute to postoperative urinary retention: (1) the patient may be nervous and self-conscious; (2) voiding may normally be difficult in the horizontal position; (3) pain in the operative wound may prevent straining; (4) the bladder reflexes may be temporarily impaired by morphine, atropine and scopolamine; (5) the lumbar and sacral nerves may have been damaged by operative trauma (e.g., abdominoperineal resection); and (6) there may be organic disease of the urinary tract or spinal cord.

Symptoms and Signs.—The patient may desire to void but cannot do so. He sometimes passes small amounts of urine but does not completely empty the bladder. There may be suprapubic pain and a feeling of "pressure." A visible or palpable suprapubic mass may be observed Unexplained restlessness is often the result of bladder distention.

Treatment.—As a prophylactic measure preoperatively, it is sometimes helpful to have the patient practice passing the urine while lying in bed. If urinary retention is to be expected, an indwelling

papaverine are indicated for their effects on autonomic and smooth muscle function, respectively. Aminophylline may also be helpful.

Anticoagulant Therapy.—*Heparin* produces a rapid and relatively easily controlled reduction in the clotting time. It must be given intravenously or subcutaneously. It is generally effective for three or four hours, and its action can be stopped with protamine sulfate (salmine sulfate). Heparin is given intravenously in doses of 50–100 mg. every four hours. If desired, it may be added to a continuous intravenous drip of glucose or saline. Ideally, the venous clotting time (Lee and White) should be determined at four hour intervals in order that the clotting time be maintained at fifteen minutes or longer. It is often sufficient to determine the clotting time once a day and to regulate the dosage accordingly. The heparin regimen may be maintained, or dicumarol may be added according to the preference of the physician. When the dicumarol effect becomes apparent (decreased prothrombin) in about forty-eight hours, the heparin may be discontinued.

Dicumarol is a hepatotoxic agent which interferes with the production of prothrombin. When used, the dosage must be carefully controlled with daily prothrombin determinations. Strict individualization of dosage is necessary. The hazards to the use of dicumarol pertain to difficulties in maintaining an optimal depressed prothrombin level without producing hemorrhage. The action of dicumarol can be counteracted by the administration of large doses of vitamin K_1, or K_1 oxide, and transfusion of fresh whole blood.

Dicumarol should be administered orally, the initial dose for the adult usually being 300 mg. The prothrombin level should be determined before the drug is given and daily thereafter. The second day, about 200 mg. should be given, and by the third day there will generally be a decrease in the prothrombin level. Depending on this response, the amount of dicumarol given thereafter will range from 0 to 150 mg. daily. The level of prothrombin to be maintained is in the range of about 20–40 per cent of normal. After the signs and symptoms of thromboembolism have cleared, it is advisable to maintain dicumarol therapy until after ambulation has been instituted. In some instances when dicumarol has been discontinued too early, venous thrombosis has recurred.

Needless to say, the patient must be protected from injury while the clotting tendency is depressed. Should operation become necessary,

Normal renal function depends on (1) the total volume of the circulating blood; (2) the rate and quantity of the blood passing through the kidney; (3) the hydrostatic and colloid osmotic pressure of the blood; (4) intrarenal, luminal and capsular pressures; and (5) the morphologic status of the glomeruli and tubules. The kidney contains two million or more nephrons, each of which is a functional unit consisting of a glomerulus and tubule. The glomerulus serves as an ultrafilter, and the tubule is designed for selective reabsorption, secretion or excretion, and synthesis and detoxification, depending on the demands of the body and the status of the internal environment.

Commonly there is more than one mechanism operating to produce anuria. The term "lower nephron nephrosis" was formerly used to denote anuria associated with certain microscopic changes largely localized in the distal convoluted tubules and the ascending limb of Henle's loop.

During the "blitz" in England (World War II) it was observed that extensive tissue damage resulting from prolonged impingement by falling timbers and masonry led to shock, hypotension, anuria, uremia and death (crush syndrome). At autopsy, focal degeneration and necrosis of the lower nephrons was observed. Other studies suggested that the degradation products of hemoglobin and myoglobin, in association with shock and alterations in blood and other body fluids, acted in combination to produce renal ischemia and anuria. The possibility that cortical ischemia resulted from reflex diversion of blood away from the cortex (glomeruli) to the medullary areas through normally little-used arteriovenous shunts was suggested by Trueta. Such a mechanism has been demonstrated in animals, but there is no certainty that it operates to produce acute renal insufficiency in man.

While the predominant histologic changes occur in the tubular cells, degenerative changes are usually widespread and involve all segments of the nephron. These changes are reversible, provided the patient survives. Tubular regeneration is generally observed within a few days and may be complete in ten days to two weeks.

Acute renal insufficiency may occur from prolonged shock, transfusion reaction (incompatible blood), extensive burning, crush injury, severe dehydration and electrolyte depletion, acute abdominal catastrophe, sulfonamide reaction and mechanical occlusion of both ureters. There is sudden onset of oliguria or anuria. Retention of metabolic waste products occurs, and symptoms of uremia soon

catheter should be introduced before operation. Postoperatively, the head rest of the bed may be elevated and the patient left alone; or if the patient's condition permits, he should be allowed to stand. Measures which are occasionally effective are: the sound of water running from a faucet in the room; the application of a hot-water bottle or a warm wet towel to the suprapubic area; a small warm enema.

Cholinomimetic drugs are not very effective. Prostigmin,® Doryl® and Urecholine® are a few of the drugs that have been used. Often they produce undesirable reactions, such as abdominal cramps, headache, flushing, hypotension or asthmatic attacks in susceptible subjects. They should not be given to patients with asthma, hyperthyroidism, bladder neck obstruction, intestinal obstruction or recent gastrointestinal resection. The action of these drugs can be counteracted by the administration of atropine.

When simple measures prove ineffective and the bladder has not been emptied for six or eight hours, the patient should be catheterized. A sterile catheter (usually No. 10 to No. 14 French) is introduced under aseptic conditions. Repeated catheterizations may be required. In the adult the bladder is overdistended if it contains more than 500 ml. If prompt return of bladder function does not follow catheterization, a retention catheter should be used until function is re-established. This may be determined by measuring the *residual urine* in the bladder The amount of urine recovered by catheterization after the patient has voided represents the residual urine. If it exceeds 150–200 ml., the indwelling catheter should be replaced or intermittent catheterization instituted. Broad-spectrum antibiotics or sulfonamides are given to minimize the possibility of urinary tract infection while the indwelling catheter is used. Bladder irrigations may also be indicated.

Acute Renal Insufficiency

Normally, a minimal urinary output of 500 cc. is required for the excretion of metabolites Lesser amounts (oliguria) or cessation of urinary excretion (anuria) results in uremia and disturbed fluid and electrolyte metabolism Although superficially the function of the kidneys is to form urine, fundamentally it is to determine the composition of the blood (Smith). In order for the kidneys to function normally certain major physiologic conditions must be fulfilled.

3. *Salt-losing diuresis* leads to severe water and salt depletion unless volume-for-volume replacement is carried out. As much as 5–10 liters of fluid may be required each twenty-four hours. The diuresis usually ceases in a few days.

If it appears unlikely that conservative measures alone will maintain the patient through the anuric phase, other measures must be considered: (1) interruption of the renal sympathetic nerve supply by splanchnic block or a decapsulation of the kidneys to effect a reduction in intrarenal pressure (these measures are rarely successful) or (2) the provision of a substitute for renal function (e.g., artificial kidney)

The artificial kidney is an external dialyzing device for clearing diffusible substances from the plasma. Heparinized blood from the patient is led through cellophane tubes immersed in a solution containing the preferred amounts of essential diffusible constituents of the plasma. The undesirable diffusible substances in the plasma are transferred through the membrane to the outside bath, and the blood is returned to the patient. Remarkable results have been reported with the artificial kidney, but its use is limited by its complexity and expense Internal dialysis is achieved by irrigating the peritoneal surfaces or the mucosal surfaces with a solution similar to that used in the artificial kidney. Metabolites such as urea and potassium diffuse into the irrigating solution and are removed. Large volumes of irrigating fluid are required. The hazard of peritonitis and the rapid deposition of fibrin on the peritoneal surfaces are drawbacks to use of peritoneal lavage

Perfusion of the small bowel or the stomach with specially prepared solutions through double-lumened tubes is a simpler and safer, but a less effective, method of dialysis.

Acute Urinary Infection

Postoperative urethritis, cystitis and pyelitis may develop from trauma, urinary retention, overdistention of the bladder and repeated catheterizations. Infection may be due to E. coli, Aerobacter aerogenes, staphylococci, streptococci or a combination of organisms. Examination of the urine will reveal pus cells, many bacteria and occasionally red blood cells.

Treatment.—The prophylactic and active measures for the treatment of acute urinary infection follow:

develop. There is slight rise in the blood pressure. Signs of heart failure appear if the circulation is overloaded or if previous heart damage exists. *Edema and acid-base imbalances can be anticipated.*

The duration of the anuria varies from three to ten days or more and depends on the severity of the renal lesion. During this period there is absorption of all or most of the glomerular filtrate through the damaged tubular membrane. The rise in urinary output on recovery coincides with re-epithelization and replacement of the tubular membrane. After tubular function has been restored, the daily urine output increases until shortly a striking diuresis appears.

TREATMENT.—Modern therapy is based on the concept that the damaged kidneys require time for cellular regeneration and functional restitution. Death results from an overwhelming irreversible renal insult or mismanagement during either the phase of anuria or the phase of diuresis. Every effort must be made to determine the cause of the anuria. If it is due to prerenal causes (hypotension, dehydration, etc.), these imbalances must be treated. If it is due to postrenal obstruction (e.g., ligation of the ureters), this must be relieved.

The major cause of the high mortality has been the infliction of a water-salt overload from too intensive parenteral fluid therapy in an effort to encourage kidney function and "wash out the toxins." If the mechanisms which lead to anuria are considered, the fallacy of this reasoning is apparent. The familiar dictum to "force fluids" has no place in the treatment of the anuric phase of acute renal insufficiency.

Management can be considered in three phases:

1. *Reaction shock* lasts a variable period of time—a few hours to several days. The immediate restoration of blood volume and blood pressure by transfusion of accurately cross-matched whole blood is essential. Plasma, plasma-expanders and other parenteral solutions may be required. Intravenous procaine, paravertebral sympathetic block and chemical sympathetic blocking agents have been used with limited success. Diuretics must not be given.

2. During the *critical anuric phase* (about seven to ten days), all fluids lost from the body should be replaced, but no excess can be given. The aim is to maintain volume, tonicity, composition and pH of the body fluids as nearly normal as possible. Generally, *it is better to keep the patient on "the dry side" rather than run the risk of overhydration and edema.* If there are no extrarenal losses, only water for metabolic needs is supplied. The oral route, if available, should be used.

remedy); or (6) procaine block or surgical crushing of the involved phrenic nerve. The latter induces temporary paralysis of the affected hemidiaphragm, which is determined preoperatively by fluoroscopy. When both leaves of the diaphragm are involved, bilateral phrenic crush may be necessary.

Acute Parotitis

Acute parotitis is an infrequent but serious postoperative complication usually encountered in very ill patients. There is a painful, localized swelling at the angle of the jaw associated with systemic signs of infection. The patient usually has poor oral hygiene and dental caries. Dehydration, malnutrition and poor oral intake after extensive abdominal operations for cancer appear to predispose to postoperative parotitis. Pyogenic organisms extend into the parotid gland by way of Stensen's duct, or are carried to it through lymphatic or vascular routes. Suppuration and destruction of the gland often follows. Acute parotitis generally occurs in the first postoperative week, and may be fatal in an already very ill patient. Bilateral involvement occurs in some cases.

Prophylactic measures should be started preoperatively and continued postoperatively. The mouth should be kept clean and moist; hydration must be maintained; and oral feedings started as early as feasible in order to encourage the salivary function.

When acute parotitis develops, warm moist compresses are applied. Early x-ray therapy to the parotid gland is quite effective. Antibiotics are also administered. If suppuration develops, surgical drainage may be necessary.

Pressure Sores

Pressure sores (bedsores [decubitus ulcers]) are the result of necrosis of the skin and deeper tissues produced by sustained local compression. They generally occur over bony prominences, such as the sacrum, iliac spines, ischial tuberosities, heels, elbows and scapulae. Cachectic, debilitated and seriously ill patients are most often affected. In the paraplegic patient, where anesthesia is associated with paralysis, bedsores are common.

Immobility and improper care of the skin, aided by heat, moisture and soiling (urine, feces, exudates), predispose to pressure sores. Improperly padded or ill-fitting casts, splints, bandages or traction

PROPHYLACTIC MEASURES:

1. Maintain an adequate urinary output.
2. Prevent overdistention of the bladder and retention of urine.
3. Observe strict asepsis when catheterization is necessary.
4. Administer antibacterial drugs if infection is likely (e.g., indwelling catheter).

ACTIVE MEASURES:

1. Antibacterial drugs, sulfisoxazole (Gantrisin®) or the broad-spectrum antibiotics, singly or in combination, according to the type of infection.
2. Bladder irrigations of sterile saline or Zephiran® 1:20,000 if there is excessive pus or debris in the urine.

In most instances, these measures will prove adequate. Bladder irrigations are probably unnecessary when a good urinary output is maintained. A more complete examination of the urinary tract is indicated if the response to treatment is unsatisfactory or organic disease is suspected.

Postoperative Hiccups

Hiccup is due to paroxysmal, intermittent diaphragmatic contraction with sudden closure of the glottis. It is a symptom, not a disease. Although usually temporary and annoying, hiccups may become protracted and exhausting. The stimulus that produces hiccup originates directly or indirectly in the phrenic nerve and may be transmitted to one or both segments of the diaphragm. The irritation may involve the efferent or afferent pathways of the phrenic nerve or its centers in the upper cervical cord.

Postoperatively, hiccup often results from mechanical or chemical irritation of the diaphragmatic peritoneum or pleura, or it may complicate intestinal obstruction, peritonitis, acute pancreatitis, intraperitoneal carcinomatosis and hepatic or renal insufficiency.

If possible, treatment is directed toward the cause. Search should be made for intraperitoneal or intrathoracic infection, acute gastric dilatation, paralytic ileus, uremia and central nervous system disorders. In most instances, hiccups will cease spontaneously or will respond to simple measures: (1) changes in position; (2) CO_2 inhalations or rebreathing the air in a paper bag; (3) gastric lavage or continuous gastric aspiration; (4) sedatives (barbiturates, bromides, morphine); (5) chloroform (a few drops placed on a lump of sugar is an old

remedy); or (6) procaine block or surgical crushing of the involved phrenic nerve. The latter induces temporary paralysis of the affected hemidiaphragm, which is determined preoperatively by fluoroscopy. When both leaves of the diaphragm are involved, bilateral phrenic crush may be necessary.

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Immobility and improper care of the skin, aided by heat, moisture and soiling (urine, feces, exudates), predispose to pressure sores. Improperly padded or ill-fitting casts, splints, bandages or traction

devices also lead to pressure necrosis, most commonly of the heel, malleoli and iliac crests.

When a pressure sore develops, the area first becomes pale, then cyanotic and blistered, and finally turns black or gangrenous. The necrotic area sloughs, and a chronic, foul-smelling, infected ulcer is formed.

Generally, the development of a decubitus ulcer can be traced to the inadequate care of the patient. All bony prominences which may be subjected to pressure must be protected. Frequent turning, meticulous cleanliness of the skin, and clean, dry, unwrinkled sheets are essential. Sponge-rubber protective pads, inflatable rubber rings and special air mattresses for paraplegics are important prophylactic items.

Splints, casts and pressure bandages must be applied with the utmost care, to avoid sustained localized pressure. When a patient complains of unexpected degrees of pain or tightness under a cast, the cast must be removed promptly and reapplied properly.

The treatment of pressure sores consists of elimination of local pressure, cleansing and débridement of the ulcer, local and systemic antibiotic therapy, and excision with closure by shifting skin flaps or grafting, depending on the size and location of the defect.

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CHAPTER 13

The Peritoneum

THE PERITONEUM lines the abdominal cavity and covers the viscera. It is composed of a single-cell mesothelial layer, the serosa, and a fibrous layer, the subserosa, which is liberally supplied with blood vessels and lymphatics. The surface area of the peritoneum approximates that of the skin. The serosa provides a smooth gliding surface for the viscera and a large absorptive surface. The peritoneal membrane is continuous except in the female, where the fimbriated ends of the fallopian tubes are a potential opening for the entry of infectious or foreign material from the exterior.

Anatomically, the peritoneal cavity consists of two parts, communicating at the epiploic foramen (foramen of Winslow): (1) the general peritoneal cavity, containing the intra-abdominal viscera, and (2) the lesser peritoneal cavity, or the omental bursa. From a clinical standpoint, the general peritoneal cavity may be divided into compartments: by the transverse mesocolon, which is attached in a transverse direction, the small-bowel mesentery, attached obliquely from the duodenojejunal ligament to the ileocecal junction, and the pelvic brim. The peritoneal compartments are:

1. *Supracolic space*—the area above the transverse mesocolon and below the diaphragm, also called the "subphrenic space."
2. *Right infracolic space*—the area inferior to the transverse mesocolon and to the right of the mesenteric attachment.
3. *Left infracolic space*—the area inferior to the transverse mesocolon and to the left of the mesenteric attachment.
4. *Pelvic space*—the area below the true pelvis and above the pelvic diaphragm.

These anatomic relationships influence the localization of pus, blood, urine, bile, pancreatic juice, gastrointestinal content, trans-

peritoneal metastases and other materials which are foreign to the peritoneal cavity.

The supracolic space deserves special attention. Many conditions involving the peritoneum originate in, or involve, this region. The several subdivisions of this compartment which are described can be best understood when demonstrated on the cadaver. Basically, the liver and its ligaments (the falciform ligament, the posterior triangular ligaments and the bare area) are interposed in the supracolic space and subdivide it in three dimensions: right and left, superior and inferior, anterior and posterior. Most commonly, abscesses localize on the right side, either above the liver and posteriorly (right superior posterior subphrenic space) or below the liver, anteriorly (right inferior anterior subphrenic space). This localization is favored by the pressure gradient (due to movement of the diaphragm) which exists between the general peritoneal cavity and the subphrenic spaces.

Fluid collections may also localize (through the influence of gravity, intraperitoneal pressure or anatomic peculiarities) in the right infracolic space, the pelvis and infrequently in the left infracolic space. The hepatorenal *pouch of Morison*, located in the right supracolic space, is of great clinical importance. It is the most dependent area to which free fluid or exudate of gastroduodenal, bile duct or pancreatic origin can gravitate.

The peritoneum has remarkable absorptive, regenerative and protective powers. The rate of absorption from all areas is relatively constant. Colloids, particulate matter and the blood are disposed of through the lymphatics or by macrophages. Crystalloids and soluble toxic products are carried off by the blood stream through subserosal capillaries. Inflammation may, at first, accelerate absorption; later there will be conglutination of fibrin, vascular thromboses and other local changes which interfere with fluid exchange and absorption.

The reparative power of the peritoneum is one factor which makes gastrointestinal operations possible. After bowel anastomosis, an early sealing-off of the serosa is accomplished by fibrin-clot formation. If the peritoneal surfaces are approximated, this process begins immediately and provides, within a short time, an insecure but leakproof union. A more substantial fibrous union occurs later; and, if conditions are right, primary healing results. The integrity of the anastomotic suture line may be endangered by improper closure, mucosal eversion, infection, hemorrhage, tension or ischemia. The presence of free peritoneal fluid may interfere with fibrin-clot formation. Depending on

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The interpretation of the signs and symptoms of abdominal origin requires an understanding of the innervation of the peritoneum. More specifically, it requires consideration of the source and type of stimulation, the pathways for transmission of pain impulses and the mechanisms of pain reference and reflex response.

The parietal peritoneum derives sensory fibers from the somatic nerves, T6 through L1. The visceral peritoneum receives sensory fibers (visceral afferent fibers) via the sympathetic chain and the splanchnic nerves. The visceral afferent fibers, the cell bodies of which are located in dorsal root ganglia from T5 to L3, are derived from and connected with the segmental somatic nerves. The essential anatomic difference between visceral and somatic fibers is their manner of distribution. Both somatic afferent and visceral afferent nerves have cell bodies in the posterior root ganglia, and both have a single neuron which extends from the receptor ending to the central nervous system without a synapse outside the spinal cord.

The sensory supply to the visceral peritoneum is sparse and localization is imperfect, as contrasted to areas of somatic innervation. The noninflamed visceral peritoneum is insensitive to the ordinary stimuli of pinch, touch, hot or cold. This is dramatically demonstrated when cherry-red cautery is used to open a colostomy without anesthesia. Visceral pain is poorly localized and diffuse; in addition, it may be felt in regions remote from the site of origin, as "referred" pain. When somatic areas are stimulated, as in parietal peritoneal irritation, localized pain and tenderness are noted. Thus, in appendicitis the initial pain resulting from appendical obstruction-distention is poorly localized and referred to the periumbilical and epigastric regions; later, when irritation of the parietal peritoneum occurs, pain and tenderness become localized to the right lower quadrant. Involuntary muscle spasm is the result of a reflex arc mediated centrally through somatic afferent nerves, the receptors of which are in the inflamed parietal peritoneum and peripherally through somatic efferent fibers to the abdominal muscles.

Classification of Peritonitis

Peritonitis is the result of mechanical, chemical or bacterial irritation, singly or in combination, producing inflammatory changes of the serous surfaces of the abdominal cavity. It may be localized, spreading or diffuse. Acute peritonitis may be classified as follows:

1. *Primary bacterial peritonitis* (uncommon)—due to the strepto-

the extent of local injury and inflammation, the healing process will progress as it does in wounds of other tissues. Regeneration of the serosal layer may be continuous or discontinuous, according to the amount of scar-tissue formation which takes place. Scarring of the peritoneum results in fibrous adhesions and the fixation of the viscera to each other or to the parietal structures. Peritoneal adhesions may often lead to functional impairment or obstruction of the gastrointestinal tract, it is true; but adhesions also have a protective localizing function as well, when intraperitoneal infection exists. Some patients exhibit a greater propensity toward forming adhesions than do others. The reasons for this are obscure. The major hazard of bowel obstruction following abdominal operations is related to the formation of fibrous adhesions. Every effort must be made to avoid trauma to the serosal surfaces and resultant peritoneal scarring.

The peritoneum has great powers of resistance to infection. Unless conditions are unusually favorable for bacterial growth, or there is continued or massive inoculation, the peritoneum can defend against and overcome most bacterial invaders. Inflammation of the peritoneum evokes an inflammatory response similar to that which occurs elsewhere in the body, modified only by the functional peculiarities of the membrane. It is an intense defense reaction, and the local peritoneal changes are designed to be protective. The systemic response reflects the intensity of the struggle between the infecting organisms and the host.

PERITONITIS

Stages of Peritonitis

When bacterial contamination of the peritoneum occurs, a local inflammatory response is initiated. First there is a violent local reaction to counteract and overcome the infection. Vascular dilatation and hyperemia with leukocyte invasion of the contaminated zone occurs. Later (perhaps within twelve to twenty-four hours) if the pathogens have not been destroyed by phagocytosis or dispersed into the oblivion of the systemic circulation, growth of the invading organism accelerates on a logarithmic scale The protective peritoneal response becomes less and less effective as vascular and lymphatic thrombosis occurs. In the fulminant case, the infection continues to extend and bacterial toxins finally produce irreparable damage of essential organ systems with peripheral vascular collapse and death. Such is the course of fatal, untreated bacterial peritonitis.

used singly or in series to describe the intensity of the pain: Awful, unbearable, stabbing and cutting are often employed with earnestness and vehemence. The pain may be described as most intense at the site of the lesion; quite as commonly it is equally distributed over the entire abdomen or may even be most intense at a site removed from the causative lesion. The verbal picture of the disease is commonly emphasized by the patient's physical appearance. Pale features, terror-stricken eyes and cold, beaded sweat covering the brow announce to the observer that the language expressed the truth. The abdominal wall is rigid. The patient may involuntarily resist examination by warding off the examiner's hand, knowing instinctively that pressure, however slight, will increase the pain. Even cursory examination shows a universal rigidity and confirms the patient's belief that pressure will increase the pain.

Only in milder cases—those in which rupture of an organ has been anticipated by a partial preliminary reaction on the part of the peritoneum, or when adhesions have so formed that but a small leak is possible—are the symptoms milder and less widely distributed. These milder cases approach the spreading type so that in the beginning one is at a loss to know which term to use and this uncertainty may extend even beyond the autopsy.

The constitutional reaction is equally striking. The pulse is rapid and forceful and may be small; it tends to become thready and also fitfully irregular as the disease progresses. The temperature may be subnormal in the beginning but has a tendency to rise, and the terminal temperature may tax the maximum registration of the clinical thermometer.

The mental state in the beginning is usually agitated as the patient attempts to acquaint the medical adviser with the intensity of his suffering. The mind remains alert, the eyes are wide and shining even at a stage when they literally sink into their sockets. Even as death approaches and the limbs are cooled by the indescribable chill of death, beginning at the extremities and gradually approaching the trunk, the mind remains clear, unmindful or fear- [illegible] The sur- [illegible]wise, and there may be a resolve to pursue a different course in the next case. Unless *the surgeon does suffer this* quickened consciousness, he is not made up of the material which is elemental to the true surgeon.

TREATMENT

Early removal or closure of a source of peritoneal contamination is a fundamental rule. Every effort must also be made at operation to remove, insofar as possible, all foreign and infectious material. At the same time, the natural barrier of the walling-off process must be respected. Irrigation and mechanical cleansing of the entire peritoneal cavity is impractical; yet much can be accomplished by repeated local suction-irrigation of areas of contamination, using physiologic saline solution.

coccus, pneumococcus, or, infrequently, the tubercle bacillus.

2. *Secondary peritonitis* (common)—due to:
   a) Diseases or injuries of the gastrointestinal tract; ruptured appendicitis, perforated peptic ulcer, ruptured diverticulitis; traumatic perforations; stab or gunshot wounds, crushing wounds, etc.; postoperative leakage; strangulating obstructions
   b) Leakage from the liver, bile passages or pancreas.
   c) Infections of the female genital organs.

Symptoms and Signs

The clinical picture is modified by the type and extent of the inciting agency, the general condition of the patient and his response to inflammation and infection.

1. Abdominal pain, which is usually severe, may be localized or diffuse.

2. Vomiting is, at first, of visceral reflex origin. It persists as a sign of peritonitis and ileus.

3. A rising pulse rate, a rising temperature and rapid respirations are signs of sepsis.

4. Diaphragmatic irritation with splinting and relative ventilatory insufficiency is responsible for shallow and rapid respirations.

5. There is direct abdominal tenderness and muscle spasm. Rebound tenderness may be noted in the absence of direct tenderness.

6. The abdomen is silent to auscultation—a most important sign of peritonism.

7. Distention of the abdomen is present in varying degrees.

8. An early sharp leukocytosis may be followed, in the fulminant case, by leukopenia, and "exhaustion" phenomena.

9. There is x-ray evidence of diffuse bowel distention. Scattered *loops of gas and fluid levels are evident in both the small and the* large bowel.

The clinical picture in advanced fulminating peritonitis with its signs of lost opportunity are unforgettable. Hertzler* has described it most graphically:

> Pain which is sudden and terrible in its onset characterizes the beginning of this type of peritonitis. All the superlative adjectives in the language are

*Hertzler, A. E.: Diseases of the peritoneum, in *Christopher's Surgery* (2d ed ; Philadelphia: W. B Saunders Company, 1939), p 1013

The main items in the care of the patient with peritonitis are the following:

1. There should be complete rest of the gastrointestinal tract: nothing by mouth; and nasogastric or long-tube suction-decompression control of the intestinal distention of paralytic ileus.

2. Parenteral fluid therapy should be instituted. Careful balance management is required. A potassium deficit may be responsible for inhibition ileus long after the peritonitis has resolved.

3. Parenteral antibiotic therapy should be given. There should be combined therapy in adequate doses, based on bacterial sensitivity tests if possible.

4. The patient should be placed in the position which is most comfortable and least restricting. Ambulation is not advisable. Fowler's position is not recommended; it contributes nothing to overcoming the peritonitis and probably predisposes to thromboembolism.

5. Hot wet packs to the abdomen may relieve pain and make the patient more comfortable, but they probably will exert little influence on the progress of the disease.

6. Morphine or related drugs should be given in small doses often enough to keep the patient comfortable. The depressant effects of large doses of morphine are to be avoided. Morphine is said to favorably influence the course of peritonitis by its metabolic effect and by a splinting action on bowel motility.

7. Drugs which stimulate bowel activity, such as neostigmine, Pitressin® and Urecholine,® have no place in treatment.

8. Oxygen therapy should be used when respiration is impaired.

The complications of peritonitis are many. It is essential that the progress of the patient be closely followed by repeatedly checking areas where trouble may arise. Common postoperative difficulties are:

1. Intraperitoneal or wound abscesses. (The locus of abscess formation may be predicted by considering the original site of peritoneal contamination and the anatomic peculiarities of the areas involved. The chief general sites in which abscesses occur are: subphrenic and subhepatic areas; the lumbar gutters, especially the right; infracolic spaces; the pelvis; and the abdominal wound itself.)
2. Paralytic ileus.
3. Mechanical intestinal obstruction.
4. Hypoventilation, atelectasis, pneumonitis and aspiration pneumonia.

Intraperitoneal drainage is indicated by the following conditions: a localized collection of pus, an area of devitalized tissue which cannot be removed, an insecure closure of a hollow organ, intraperitoneal leakage of body fluids (blood, bile, etc.) which cannot be controlled at the time of operation and extensive retroperitoneal contamination.

A drain does not provide automatically for complete evacuation of *body fluids or exudates but functions according to the physical* laws which govern the movement of all liquids. By its presence, as a foreign body, a drain may interfere with normal peritoneal absorption or even may increase peritoneal exudation. Eventually, all drains become sealed off from the peritoneal cavity by exudate and may function as plugs instead of vents. Drains also incite the formation of peritoneal adhesions. Stiff or hard rubber drains in contact with soft tissues are dangerous. They may cause pressure necrosis, hemor-*rhage and fistula formation An abdominal drain brought through* a wound may weaken the abdominal wall and increase the chance of infection and wound disruption. Properly used, however, drains serve an important function, and there probably is merit to the old dictum "When in doubt, drain."

At present there is little indication for the intraperitoneal use of antibiotics The prophylactic use of "intestinal" sulfonamides and broad-spectrum antibiotics in bowel preparation, the occasional instillation into the bowel at the time of operation of an antibiotic such as neomycin, and the maintenance of high systemic concentrations of penicillin, streptomycin and broad-spectrum antibiotics will give a high degree of protection against peritoneal infection.

Attention must be given to the patient's preoperative state, and all necessary measures taken to improve his condition. This usually requires the correction of fluid-electrolyte, acid-base and blood deficits, decompression of the gastrointestinal tract and institution of vigorous antibiotic therapy. Often the course to be followed will be clearly indicated by the circumstances leading to the peritonitis. For example, if the patient has early peritonitis due to a gunshot wound of the abdomen, there is no choice but to operate; if diffuse peritonitis is due to ruptured appendicitis or ruptured peptic ulcer, early operation is indicated, but if peritonitis is due to pelvic inflammatory disease, nonoperative treatment is usually advisable. The longer in time the duration of the peritonitis, the more difficult it is to judge the proper timing of operation. Individualization, therefore, must be the basis *for these decisions.*

The main items in the care of the patient with peritonitis are the following:

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2. Paralytic ileus.
3. Mechanical intestinal obstruction.
4. Hypoventilation, atelectasis, pneumonitis and aspiration pneumonia.

5. Thromboembolic disease (phlebothrombosis).
6. Wound-healing complications: infection, disruption, evisceration and hernia formation.

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CHAPTER 14

The Upper Alimentary Canal

THE UPPER alimentary canal, extending from the oral cavity to the duodenal papilla (including the liver, bile passages, and pancreas) is derived from the foregut embryologically. It is concerned with the conduction and mixing of material entering it and with the elaboration and secretion of digestive juices.

The organs of the upper canal include: the esophagus, stomach and first part of the duodenum, each of which have certain well-known structural similarities and dissimilarities. They are similar in that they are all hollow organs, lined with mucous membrane and able to propel their contents by virtue of a smooth muscle coat. They are all richly supplied with arteries, veins, lymphatics and autonomic nerves. The arterial supply of the stomach and proximal duodenum (as well as the liver and most of the pancreas) is derived from the celiac axis. The venous drainage of all the abdominal viscera is to the portal system. The lymphatic drainage is through channels which follow the arteries. The nerve supply is derived from splanchnic fibers which come to the foregut originally from segments T5 to T9 and from parasympathetic (vagal) fibers. In common with the entire alimentary canal, this segment possesses an intrinsic nervous mechanism consisting of the submucosal plexus (Meissner) and the intermuscular plexus (Auerbach).

THE ESOPHAGUS

The esophagus begins at the lower end of the pharynx, traverses the neck, mediastinum and diaphragm and becomes continuous with

the stomach at the cardio-esophageal junction. Throughout its course it lies in close relationship to the trachea, thyroid gland, aorta, root of the lung, heart, vagus nerves, thoracic duct, spinal column and diaphragm. The lymphatics of the upper esophagus drain to the cervical nodes, those of the midesophagus to the mediastinal nodes, and those of the lower esophagus to the mediastinal and upper gastric nodes. The esophagus has no outer serous covering.

The foregoing anatomic features have important clinical implications. For example: the surgical approach to the esophagus is generally difficult because of the location and proximity of the esophagus to

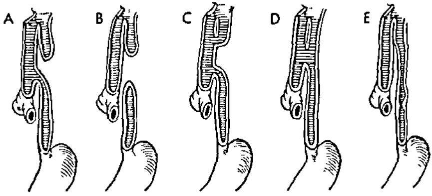

FIG. 56—Tracheo-esophageal anomalies of the newborn. *A*, most common type: upper segment of the esophagus ends blindly; lower segment communicates with the posterior surface of the trachea. *B*, same as above, but lower segment of the esophagus does not communicate with the trachea. *C*, both upper and lower segments enter the trachea *D*, segments are continuous, but there is a tracheo-esophageal communication *E*, esophagus is stenotic in its midportion.

other important structures, early extraesophageal spread of infection and cancer to inaccessible areas occurs commonly; and operative and traumatic wounds of the esophagus are more predisposed to leakage than similar wounds of the abdominal viscera because the esophagus does not have a self-sealing serous coat.

Diseases and injuries of the esophagus are infrequent in clinical practice. The student should have a working knowledge of the more common conditions, which include those of (*a*) congenital and acquired origin, (*b*) traumatic origin, (*c*) inflammatory origin and (*d*) neoplastic origin. Only a few aspects of these conditions will be considered here.

Congenital atresia of the esophagus and tracheo-esophageal fistula (Fig 56) are of developmental origin and are the result of a faulty separation of the respiratory passages from the primitive foregut.

Several anatomic variations are encountered, but the most common defect (about 90 per cent) consists of a short segment of upper esophagus, which ends as a blind pouch, and a longer distal segment, which at its upper end communicates with the trachea or a main bronchus. The lower end of the distal segment joins the stomach normally (Fig. 56, *A*).

The symptoms are the result of esophageal obstruction and reflux of fluid and air through the fistula. There is dyspnea, cyanosis and an inability to take food without immediate regurgitation. The diagnosis is confirmed by the inability to pass a catheter through the esophagus into the stomach and by the demonstration of a blind esophageal pouch by x-ray. Iodized oil, *not barium*, must be used. Urgent surgical treatment is necessary. Through an incision in the right side of the chest the tracheo-esophageal fistula is identified and closed, and the two segments of the esophagus are united by suture.

Varices of the esophagus are most commonly the result of portal hypertension with portal cirrhosis. The varicosities tend to ulcerate and bleed massively. This condition is discussed in Chapter 18, on The Liver.

Diverticuli of the esophagus occur principally in two areas—the neck and the supradiaphragmatic region. The cervical or hypopharyngeal type (Zenker's diverticulum, Fig. 57) is most common. Diverticuli regularly appear on the posterior surface of the pharynx through an intermuscular defect between the inferior constrictor and the cricopharyngeus muscles. As the diverticulum enlarges, it commonly passes to the left side of the neck, where it may be visible and palpable. The symptoms of cervical diverticuli include: dysphagia, regurgitation, gurgling noises in the neck with swallowing and, sometimes, intermittent localized swelling. The diagnosis is established by x-ray studies and may be confirmed by esophagoscopy. Treatment consists of operative removal and closure of the opening in the esophagus.

Injuries of the esophagus most often result from penetrating wounds or instrumentation (e.g., esophagoscopy). Spontaneous rupture of the esophagus is rare. The diagnosis is made by x-ray or esophagoscopy. Small perforations may seal off and heal spontaneously, but the danger of leakage and mediastinitis must always be considered. Early closure of perforations is usually indicated.

The esophagus can also be damaged by strong chemicals (e.g., lye) or foreign bodies.

Inflammation of the esophagus can occur after all forms of trauma.

FIG 57 (*left*).—Diverticulum of the esophagus (hypopharynx). X-ray taken in lateral projection. Note that the sac passes behind the esophagus and into the thoracic inlet In the anteroposterior projection it was found to pass to the left side of the neck, which is the usual location The patient was cured by operative removal of the sac and closure of its orifice into the esophagus.

FIG 58 (*right*) —Carcinoma of the esophagus with partial obstruction. Note the irregularity and narrowing of the esophageal lumen produced by the lesion The condition was found to be incurable at operation

Spontaneous ulceration of the lower esophagus, presumably due to acid-peptic digestion, may clinically simulate or be associated with peptic ulcer of the stomach or duodenum. The treatment of this condition includes those measures regularly prescribed for peptic ulcer, including partial resection of the stomach in some cases.

Benign tumors of the esophagus are rare, while *malignant tumors* are common Cancer of the esophagus occurs most frequently in males beyond middle life. The onset of this condition is insidious, consisting

at first only of slight discomfort on swallowing. Later there is difficulty in swallowing many solid foods, and finally only liquids will pass. Unfortunately for the patient, he has no pain and generally does not seek medical attention until the disease has progressed to an incurable state. The diagnosis of esophageal cancer should be suspected in all patients who complain of "food sticking." The investigation includes x-ray studies of the esophagus with barium (esophagram, Fig. 58) and esophagoscopy with biopsy.

Carcinoma is most frequent in the lower third of the esophagus, but it also occurs in the middle, upper and cervical segments. The tumor is commonly a squamous cell carcinoma, less commonly an adenocarcinoma. At operation, metastases to the mediastinal lymph nodes and invasion of surrounding structures is encountered in a high percentage of patients. Surgical treatment consists in resection of the lower esophagus with the tumor and uniting the proximal esophagus with the stomach. In order to make such an anastomosis, it is necessary to displace the stomach into the thorax. The operative mortality is approximately 20 per cent, and the five year survival rate is low.

Palliative treatment of esophageal carcinoma includes: introduction of a plastic tube into the lumen and through the narrowed area produced by the tumor by open operation; establishment of gastrostomy for feeding purposes; and external irradiation.

THE STOMACH

The stomach is a J-shaped pouch between the esophagus and the duodenum. The inlet of the stomach is the site of the cardio-esophageal sphincter, and the outlet is demarcated by the pyloric sphincter. The superior segment of the stomach or the vertical part of the "J" is the *cardia*. The cardia consists of two parts: the *fundus* above, and the *body* below. The inferior segment of the stomach, or the horizontal part of the "J," includes the *antrum* and the *pyloric canal*. The junction of the cardia and the antrum on the lesser curvature of the stomach is the site of the *incisura angularis*. The shape, size and position of the stomach vary greatly from individual to individual. These variations can best be appreciated by viewing several sets of x-ray films of the stomach.

The stomach wall consists of four layers: the serosa, muscular coat, submucosa, and mucous membrane. In the gastric glands the mucous membrane contains four types of cells. The body chief cells, or the

zymogenic cells, secrete pepsin; the parietal, or oxyntic, cells produce hydrochloric acid, the mucous neck glands secrete mucus; and the argentaffine cells probably produce an internal secretion, serotonin.

The acid-producing parietal glands are found in greatest number

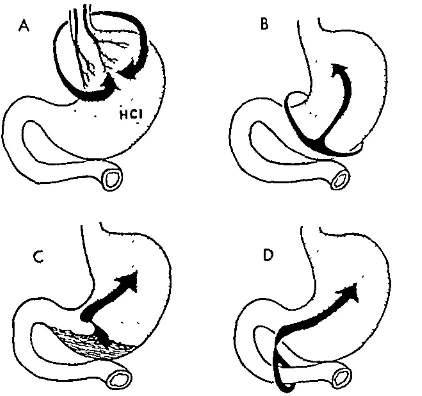

FIG. 59.—The mechanisms of acid gastric secretion. *A*, cephalic phase, under neurogenic control through the vagus nerves. *B*, gastric phase; hormonal stimulation is the result of antral production of gastrin. *C*, gastric phase, direct stimulation by presence of food in the stomach. *D*, enteric phase, controlled by enterogastrone produced by the duodenum and jejunum.

in the central portion of the stomach. They are less numerous in the region of the incisura angularis and gastric fundus and practically absent in the distal pyloric area.

Gastric secretion occurs in three phases (Fig. 59): the cephalic, or nervous; the gastric, or hormonal, and the intestinal The cephalic is initiated by the sight, smell, taste, chewing or thought of food. The secretory reflex is mediated over the vagus nerves and results in a secretion which is rich in acid, pepsin and chlorides Section of the vagus nerves interrupts this reflex.

The gastric phase of secretion results from both hormonal (gastrin)

and mechanical stimulation. Gastrin is liberated in the antrum in response to the presence of food and to distention of the antrum. This hormone in turn acts on the mucosa to produce acid secretion. Histamine is used clinically to provoke acid gastric secretion, and this test probably reflects the hormonal phase of gastric secretion. Removal of the antral segment reduces or eliminates this phase of gastric secretion.

The intestinal phase of gastric secretion appears to be due to the action of secretagogues on the intestinal mucosa, which results in the liberation of a humoral agent which behaves much as does gastrin.

There appears to be a resting or interdigestive phase of gastric secretion which is also under neurogenic control. This has been demonstrated in ulcer and nonulcer patients by comparing the total night secretion of these groups. The volume of night secretion is generally greater in ulcer patients than in normal individuals.

Gastric juice contains hydrochloric acid, pepsin which is derived from pepsinogen by the action of hydrochloric acid, and mucus. It is known that mucus has a mucosal protective function, with respect to acid-peptic digestion. But whether or not the enzyme, lysozyme, which has the ability to dissolve mucus, plays a significant role in the pathogenesis of peptic ulcer has not been established.

The duodenum contains bile, pancreatic juice and small-bowel secretions. The reaction of this mixture is alkaline. Under normal conditions the acid gastric chyme is neutralized when it passes into the duodenum. This change in reaction is an important normal protective mechanism against ulceration of the duodenum.

DISEASES OF THE STOMACH AND DUODENUM

A variety of pathologic conditions occur in these organs. Many are so-called "medical problems" and are dealt with in medical works. Peptic ulcer and cancer of the stomach are of combined medical and surgical interest and illustrate many of the problems with which the surgeon is concerned.

Peptic Ulcer

From a clinical viewpoint, gastric and duodenal ulcers have many points of similarity, including pathogenesis, signs and symptoms, methods of diagnosis, complications and surgical treatment. For this

reason, they will be considered together in the following discussion.

Peptic ulcers develop only in the mucous membrane of certain areas which are exposed to acid and pepsin. Most commonly they are located along the lesser curvature of the stomach or the first part of the duodenum. Less often they appear in other areas of the stomach (antrum, body, fundus, etc.); and only occasionally in the lower esophagus, second portion of the duodenum, Meckel's diverticulum

FIG 60.—Bleeding gastric ulcer The distal portion of the stomach was resected for massive hemorrhage from a benign peptic ulcer. The specimen has been turned inside out to show the ulcer. Note the medium-sized artery in the ulcer crater.

containing ectopic gastric glands, or the jejunum after gastrojejunostomy.

Peptic ulcers (Fig 60) are punched-out excavations with slightly overhanging margins and granulating bases. They exhibit a variable degree of surrounding inflammatory reaction, scarring, deformity and regional lymph node hyperplasia. They are usually single and range from a few millimeters to 2 cm. or more in diameter. Occasionally, several ulcers occur in the same area.

The ulceration may be confined to the mucosa or may penetrate the entire thickness of the gastric or duodenal wall. When sudden

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The ulceration may be confined to the mucosa or may penetrate the entire thickness of the gastric or duodenal wall. When sudden

complete penetration occurs, irritant gastric or duodenal content leaks into the peritoneal cavity. Slow penetration and perforation activates defense mechanisms which tend to seal the opening and localize the inflammation. When penetration occurs in a retroperitoneal area (e.g., posterior wall of the duodenum), peritoneal soiling does not occur.

The digestive action of acid and pepsin often causes the erosion of blood vessels and hemorrhage. Erosion of veins, capillaries and [illegible] severity.

[illegible]

The clinical course in peptic ulceration is characteristically one of recurrences and remissions. With each episode, more scar tissue is produced and irregularities of the gastric or duodenal wall appear. The resulting contraction or deformity may be sufficient to ultimately cause partial or complete obstruction. A variable degree of superimposed spasm and edema may in itself cause partial obstruction.

Acid gastric juice is necessary for the development and persistence of peptic ulcer. The actual cause or causes of peptic ulcer are unknown. In the stomach there appears to be a breakdown in the normal resistance to tissue digestion, which may be due to a diminished protective action of mucus or to local cellular changes related to disturbances in vasomotor control. In the duodenum, exposure to excessive acid gastric secretion, rather than a decreased efficiency of local protective mechanisms, is likely.

That neuroendocrine factors are also involved is established. Peptic ulcer is a disease of civilization. The relationship between the emotions, the antonomic nervous system and the motor and secretory activity of the stomach is well known. Prolonged emotional turmoil from any cause results in disturbed gastric function, vascular mucosal engorgement and increased susceptibility of the mucosa to digestion.

The significance of the hormones, in this respect, is indicated by studies on the relationship between "stress," gastric secretion and adrenocorticoid activity. Superficial ulcerations of the upper gastrointestinal membrane are common under conditions of stress. Cortisone and ACTH regularly augment gastric secretion and lead to serious complications when administered to patients with ulcer, or they may precipitate ulceration in those who are predisposed to this disease.

Symptoms and Signs.—The ulcer syndrome is characterized by pain, punctuality and periodicity. Sometimes there is little more than a sense of fulness and discomfort after eating; but more often the

patient complains of a burning, gnawing, nonradiating abdominal pain with epigastric tenderness. The pain occurs one to three hours after meals and frequently in the early morning hours. It is characteristically relieved by ingestion of food or alkalis.

Vomiting is uncommon except when there is partial obstruction. If present, the vomitus may contain "coffee ground"-appearing material

FIG 61.—Roentgenogram of the barium-filled stomach showing an ulcer niche on the lesser curvature near the incisura angularis

or flecks of clotted blood. Ordinarily, the stools appear normal but are found to contain occult blood. With more severe bleeding the stools become "tarry" or grossly bloody.

The physical examination is generally unrevealing except for localized upper abdominal tenderness. Weight loss and anemia are infrequent. Gastric secretory studies usually reveal hyperacidity in duodenal ulcer and normal or hypoacidity in gastric ulcer.

The radiographic signs of ulcer are (1) an ulcer niche seen on the x-ray film (Fig. 61), (2) localized tenderness over the suspected ulcer at fluoroscopy, (3) hypertonicity and hypermotility of the stomach and (4) pylorospasm with duodenal deformity.

Gastroscopy is often useful for the differentiation of benign and malignant gastric lesions and the diagnosis of gastritis and gastric lymphoma. Its value is limited by the fact that in the inspection of the interior surface of the stomach there are several "blind spots." Gastroscopy is of no value in duodenal ulcer.

The complications of peptic ulcer (Fig. 62) comprise the chief indications for surgical treatment. They are: acute perforation, hemorrhage, pyloric obstruction, intractability, and suspected malignant disease in the case of gastric ulcers only.

PERFORATION.—This is quite frequently a catastrophic complication. The perforation is usually located on the anterior gastric or duodenal wall near the pyloric ring. At operation it is found to be a cleanly punched-out defect from which gastric and/or duodenal contents are leaking into the peritoneal cavity. There is rapid and diffuse soiling of the peritoneum with this highly irritating liquid mixture. The immediate local reaction is a chemical peritonitis; later the peritonitis may become infectious. The systemic reaction tends to be severe, and decompensation of the homeostatic mechanisms with shock often appear.

The signs of ruptured ulcer are usually quite characteristic. The patient may have a known history of peptic ulceration. Often perforation will follow a bout of ulcer distress. The onset of perforation is heralded by sudden upper abdominal pain, which becomes severe and diffuse as the free fluid spreads over the peritoneal surface. Pain in the right shoulder is commonly observed and results from diaphragmatic irritation. Pain in the right lower quadrant may occur from the gravitation of fluid along the right lumbar gutter, in which case the clinical picture may simulate acute appendicitis. Vomiting is uncommon. The picture may be that of impending shock, manifested by cold skin, sweating, fast pulse and decreased blood pressure. Later, shock becomes established unless treatment is given. The abdominal wall is exquisitely tender and boardlike. The area of normal liver dulness is replaced by resonance to percussion by virtue of free air above the liver. The bowel sounds are hypoactive or absent. The scout films of the abdomen confirm the presence of pneumoperitoneum.

Perforated peptic ulcer is a surgical condition, and closure of the

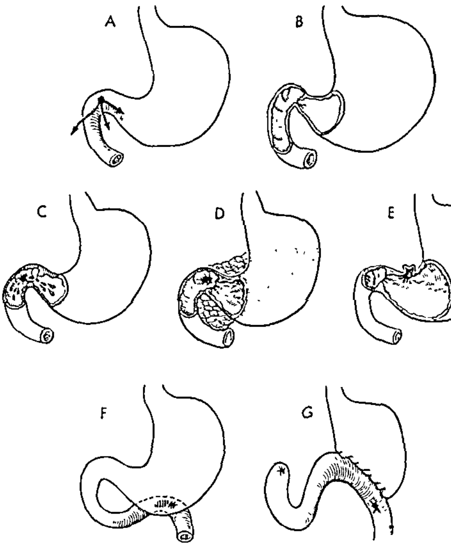

FIG 62.—Common complications of peptic ulcer which require surgical treatment *A*, acute perforation with peritonitis. *B*, obstruction to the pyloric outlet. *C*, hemorrhage, recurrent or massive *D*, intractability (sometimes the result of penetration into the pancreas). *E*, suspected malignant changes in gastric ulcers. *F*, jejunal (marginal or stomal) ulceration following gastrojejunostomy. *G*, jejunal (marginal or stomal) ulceration following partial gastric resection

ulcer should be accomplished at the earliest possible moment. The morbidity and mortality in this condition is related directly to delay. A short initial period of preoperative treatment, not exceeding two or three hours, is permissible, after which simple closure is carried out. All accessible free intraperitoneal fluid and foreign material should be removed. This will decrease the chances of postoperative intraperitoneal abscesses.

Recovery from a ruptured ulcer is usually uncomplicated, provided that early surgical treatment is applied. The postoperative management is similar to that of patients with peritonitis of any type.

HEMORRHAGE.—Bleeding results from vascular erosion. Massive bleeding often comes from branches of the gastroduodenal artery or the right gastric artery. Massive hemorrhage is always a life-threatening complication. If the physician understands the implications of this condition, he will be in a position to deal with it, as well as with less severe bleeding problems.

Sometimes the patient will have had a previous bleeding episode and will recognize what is happening. There may be a history of a pre-existing peptic ulcer. The patient who has a typical "bathroom hemorrhage" feels the urge to defecate and faints while in the bathroom. He may pass one or more black or bloody stools, become nauseated and vomit blood-containing fluid. The physician is often able to make the diagnosis over the telephone. This condition is a real emergency.

While many patients with bleeding peptic ulcer can be managed by nonoperative measures, certain patients require immediate operation to stop bleeding and to save life; others require operation after the cessation of the bleeding, to prevent recurrence. The criteria for the selection of patients for surgical treatment cannot be rigid. Each problem must be judged according to the individual circumstances and in light of the known increased risk when certain conditions which adversely affect the outlook exist. These include the following:

1. The age of the patient is of importance, since the mortality increases with age. In general, the younger the patient the better he is able to tolerate hemorrhage. After the age of forty-five the risk increases, but patients younger than this also die from bleeding ulcer. The relationship between age, severity of hemorrhage and prognosis is ascribed to changes in the vascular system (arteriosclerosis) and to thickening and fixation of vessels in the ulcer bed. These changes are most marked in patients with the scarring of chronic recurrent ulcera-

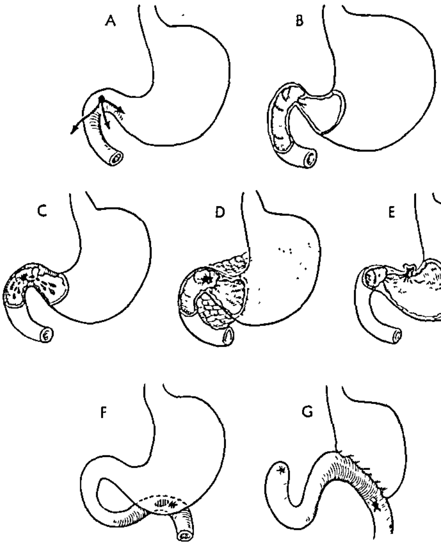

FIG. 62.—Common complications of peptic ulcer which require surgical treatment. *A*, acute perforation with peritonitis. *B*, obstruction to the pyloric outlet. *C*, hemorrhage, recurrent or massive. *D*, intractability (sometimes the result of penetration into the pancreas). *E*, suspected malignant changes in gastric ulcers. *F*, jejunal (marginal or stomal) ulceration following gastrojejunostomy. *G*, jejunal (marginal or stomal) ulceration following partial gastric resection.

ing the ulcer, if possible. When, at operation, the critical condition of the patient precludes resection, simple measures for the control of bleeding, such as transfixation of the vessel, may be all that can be done.

Obstruction.—Long-standing chronic peptic ulcer of the gastric outlet and duodenum may, by virtue of scarring and deformity, lead to the mechanical blockage of the lumen. Often there is a superimposed element of edema with muscle spasm which results in almost complete obstruction. The stomach then gradually becomes dilated and hypertrophied. *Retention of food and of gastric secretion occurs,* and chronic gastritis develops.

The patient complains of chronic indigestion, fulness, night pain, vomiting, inability to eat solid foods, loss of appetite, weight loss and weakness. His condition deteriorates progressively; and signs of dehydration, alkalosis, anemia, hypovolemia, hypoproteinemia and chronic malnutrition are often evident to the physician.

The diagnosis of pyloric obstruction is made on the basis of the clinical findings and the x-ray studies. It may be impossible, however, to differentiate between a benign and a malignant obstruction.

Patients with prolonged pyloric obstruction require a period of decompression and mechanical cleansing of the stomach, as well as nutritional restoration, before surgical treatment can be undertaken. Often the *patient's symptoms clear, and partial relief of the obstruction* will allow him to take an ulcer diet or formula feedings. The preoperative regimen requires a few days to a few weeks, during which the patient's general condition rapidly improves and he soon becomes a satisfactory surgical risk. The operations for outlet obstruction include: partial gastric resection, anastomosis of the stomach to the jejunum and enlargement of the gastroduodenal outlet (pyloroplasty).

Intractability.—Peptic ulcer is said to be intractable when improvement does not follow a prolonged and exacting period of medical treatment. That is, the symptoms persist, the ulcer fails to heal and the patient remains disabled in spite of strict dietary, antacid and anticholinergic therapy. This type of management is best carried out in a hospital, usually for a period of not less than three weeks. In this regard, the *physician must make sure that it is the ulcer,* rather than the patient, which is intractable. Generally speaking, patients who are resistant and unco-operative are poor candidates for either medical or surgical treatment.

Surgical treatment of intractable chronic peptic ulcer includes

tion, which is most likely to be encountered in those of advanced age.

2. The vomiting of blood has more serious significance than blood in the stools alone. It may indicate gastric or esophageal ulceration and rapid hemorrhage.

3. Recurrence of hemorrhage while the patient is under treatment for peptic ulcer is always a serious sign.

4. Associated diseases, such as cardiac failure, hypertension and cirrhosis of the liver, always make the outlook darker.

5. Failure to respond after restoration of the blood volume is an important factor. In borderline cases, if a "test of transfusion," amounting to 1,500 or 2,000 ml. of whole blood in twenty-four hours, does not stabilize the patient's condition, the implication is that there is continued bleeding. Surgical intervention must be considered.

The cause of upper gastrointestinal bleeding may be difficult to determine. Peptic ulceration (duodenal and gastric) is responsible for about 75 per cent of cases; and ruptured esophageal varices, gastritis and malignant conditions account for about 20 per cent. The history, physical findings and the character and amount of shed blood may suggest its origin. Clotted or unclotted blood in large-appearing amounts from the mouth indicates rapid hemorrhage from the upper alimentary tube, usually from above the pyloric sphincter area. Black or hemolyzed blood in the vomitus may come from the stomach or duodenum, dark red clotted and unclotted blood, appearing from the rectum, can originate from a segment of the alimentary tract, including the upper portion, when bleeding is brisk and conduction is rapid. Black liquid (tarry) stools are commonly the result of upper-tract bleeding and can result from the loss of only 75 ml. of blood into the stomach or duodenum.

Every effort must be made to localize the site of bleeding when operative treatment is being considered. Upper gastrointestinal x-ray examination may be necessary to rule out esophageal varices and to demonstrate the presence of an ulcer. Esophagoscopic examination may also be indicated. When ruptured esophageal varices are the probable cause of bleeding, nonoperative measures are generally indicated, including the Blakemore-Sengstaken tube. Likewise, if the patient with bleeding ulcer stops bleeding and shows signs of improvement after the institution of supportive treatment, the decision may be made in favor of continuing nonoperative management.

The surgical treatment of hemorrhage from peptic ulcer consists in the control of bleeding by partial resection of the stomach, includ-

ing the ulcer, if possible. When, at operation, the critical condition of the patient precludes resection, simple measures for the control of bleeding, such as transfixation of the vessel, may be all that can be done.

OBSTRUCTION.—Long-standing chronic peptic ulcer of the gastric outlet and duodenum may, by virtue of scarring and deformity, lead to the mechanical blockage of the lumen. Often there is a superimposed element of edema with muscle spasm which results in almost complete obstruction. The stomach then gradually becomes dilated and hypertrophied. Retention of food and of gastric secretion occurs, and chronic gastritis develops.

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INTRACTABILITY.—Peptic ulcer is said to be intractable when improvement does not follow a prolonged and exacting period of medical treatment. That is, the symptoms persist, the ulcer fails to heal and the patient remains disabled in spite of strict dietary, antacid and anticholinergic therapy. This type of management is best carried out in a hospital, usually for a period of not less than three weeks. In this regard, the physician must make sure that it is the ulcer, rather than the patient, which is intractable. Generally speaking, patients who are resistant and unco-operative are poor candidates for either medical or surgical treatment.

Surgical treatment of intractable chronic peptic ulcer includes

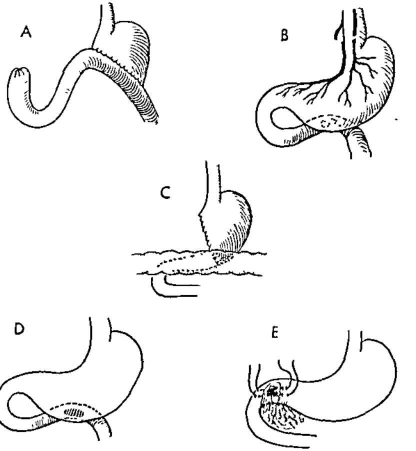

FIG. 63.—Common surgical procedures used in the treatment of peptic ulcer. *A*, partial gastric resection, Polya's modification of Billroth II type (end-to-side gastrojejunostomy). *B*, vagotomy and posterior gastrojejunostomy. *C*, partial gastric resection, Billroth I type (end-to-end gastroduodenostomy). The position of the transverse colon is indicated. *D*, posterior gastrojejunostomy. *E*, closure of perforated ulcer by suturing a tab of omentum over the opening.

partial gastric resection and, in some clinics, vagotomy and gastroenterostomy.

SUSPECTED GASTRIC CANCER.—Cancer of the stomach can masquerade as gastric ulcer, and in rare instances, benign gastric ulcers are believed to become cancerous. About 10 per cent of the patients operated on for apparently benign ulcers are found to have histologically malignant changes in the ulcer. Furthermore, it is often impossible to distinguish between malignant and benign ulcers by gross examination of the excised specimen. It follows, therefore, that there are no clinical means by which the differentiation can be made with

certainty, short of biopsy, and that the clinician always must consider the possibility of gastric cancer when dealing with gastric ulceration.

The history, gastric secretion, x-ray findings, and sometimes the gastroscopic studies, will indicate the most likely diagnosis. If the evidence favors benign gastric ulcer, there are two alternatives. The physician may elect to place the patient on strict medical treatment for a period of three or four weeks and then to review the situation completely, including the x-ray studies. This is a form of diagnosis by observing the response to treatment. At the end of this period, a definite diagnosis should be made and medical treatment continued or surgical treatment advised. The second, more direct approach consists in advising operation for all patients with gastric ulcers. There is much to be said in favor of early operation, which serves as a combined diagnostic and therapeutic measure

SURGICAL TREATMENT.—Most patients with peptic ulcer are treated by medical measures. Every reasonable effort should be made to heal the ulcer, to relieve the symptoms, to restore normal activity and to prevent recurrences by nonsurgical means, whenever possible. Failure to achieve these objectives or complications of ulcer call for surgical treatment (Fig. 63). When indicated, surgical treatment will, in a high percentage of patients, accomplish these objectives; but it is by no means a cure-all

Currently, two basic types of surgical operations are used in the treatment of peptic ulcer. The first consists of partial removal (two thirds to three fourths of the distal stomach) closure of the duodenum and re-establishment of continuity by uniting the divided end of the stomach and the upper jejunum.* This procedure has many modifications and eponymic labels, but the original procedure is known as "Polya's operation." Its effectiveness in the treatment of peptic ulcer lies in the fact that the antral source of gastrin is eliminated, the acid-secreting membrane is reduced and, in most instances, the ulcer is removed. This operation (partial gastric resection) is widely used.

The second operation, which is less widely used, consists in the division of both vagus nerves (vagotomy) at or above the esophageal hiatus and the establishment of an opening between the side of the stomach and the side of the jejunum (gastrojejunostomy) to prevent

*An alternative method for re-establishment of continuity is direct suture of the divided end of the stomach to the divided end of the duodenum (Billroth I operation). The amount of the stomach removed is the same (Fig. 63, C).

gastric retention from disturbed motor activity postoperatively.* This operation effects a reduction in the cephalic phase of gastric secretion, which leads to healing of the ulcer.

The mortality from gastric operations is low, averaging about 3 per cent. The common postoperative complications include: obstruction at the anastomosis and gastric retention, leakage from the duodenal stump closure, intraperitoneal abscesses, wound disruption, pneumonitis and phlebothrombosis.

The long-term results of treatment are satisfactory in about 85 per cent of cases after both partial gastric resection and vagotomy with gastroenterostomy. It should be pointed out that even after operation the patient with the ulcer diathesis is, to some degree, more predisposed to recurrent related difficulties than the normal person.

Late complications which may follow partial gastric resection include: the postgastrectomy syndrome, stomal or marginal ulceration and failure to gain weight.

The postgastrectomy syndrome, sometimes called the "dumping syndrome," is characterized by epigastric distress, lightheadedness, weakness, cold sweats and palpitation, appearing shortly after the ingestion of food. These manifestations have been variously ascribed to (1) rapid gastric emptying, (2) jejunal or duodenal distention, (3) sudden shifts in plasma volume due to the movement of fluid from the vascular compartment into the bowel, (4) disturbances in glucose metabolism and (5) disturbances in adrenocorticoid function. The symptoms are less severe or disappear if frequent small feedings, containing minimal fluid and carbohydrate, is prescribed and if the patient lies down after eating. Over a period of weeks or months the symptoms tend to disappear unless there is a mechanical cause for the trouble.

Stomal or marginal ulceration occasionally develops in the jejunum near the anastomosis as a result of acid-peptic digestion. It is most likely to occur after simple gastroenterostomy and rarely after partial gastric resection. The ulcer may appear a few weeks after operation but is usually not seen until months or years have passed. The new ulcer has all the characteristics of the original ulcer except for its location. Ulcer pain is often severe. Bleeding is common. Perforation may occur into the free peritoneal cavity or sometimes into the nearby transverse colon. The latter results in a gastrojejunocolic fistula, which is an extremely serious complication of stomal ulcer. The diagnosis

* Pyloroplasty rather than gastrojejunostomy is preferred by some surgeons.

of stomal ulceration and gastrojejunocolic fistula is made by x-ray. Treatment is surgical.

Failure to gain normal weight is common after partial gastrectomy. This may be due to inadequate intake or inadequate utilization of food. There is evidence to indicate that the greater the segment of stomach removed, the greater will be the inability to gain. For this reason, some surgeons believe that resection should, in some degree, be tailored to the patient's existing nutritional status. That is, chronically thin patients should be subjected to more conservative gastric resections than well-nourished persons. In most instances, however, patients who fail to gain to normal weight postoperatively can be made to gain under rigid dietary management.

Cancer of the Stomach

According to Maes*:

> The only safe plan is to regard as cancer any indigestion, with or without symptoms, which appears after middle life in a previously well person; to regard as cancer any acute digestive disturbances, in this period, which are superimposed upon chronic digestive disturbances and which do not respond to routine measures; to regard as cancer or highly suspicious of it, vague general symptoms of fatigue, malaise, mental indifference, insomnia, etc., even though associated gastric disturbances are lacking; to continue to regard as cancer any of those clinical syndromes until it is proved beyond the shadow of a doubt not to be cancer; and to resort without delay to exploratory laparotomy, if the diagnosis cannot be made otherwise. In malignant disease the certainty of diagnosis is frequently also the certainty of death.

Clinical Considerations.—Cancer of the stomach has long been a foremost medical problem and threatens to become even more important as the number of people living to advanced age increases. In the United States, there are about 40,000 deaths a year from cancer of the stomach. The cure rate in this disease has always been low (about 10 per cent). There appears to be some improvement in the survival statistics in recent years, but there is great need for more improvement. To this end, there is need for continued public emphasis on the importance of prompt medical investigation of digestive complaints.

Gastric cancer is more frequent in the male than the female, the ratio being about 2:1. It appears with greatest frequency after the

*Maes, U.: *Carcinoma of the stomach*, New Orleans M. & S. J. 90.584, 1937-1938.

age of forty but is not rare in younger people. Most often it develops in the absence of pre-existing gastric disease. However, a few conditions are regarded as precursors of gastric cancer. These include:

Gastric Ulcer.—*Probably less than 5 per cent of the gastric ulcers* undergo malignant degeneration. Of greater importance is the fact that benign and malignant gastric ulcers are often impossible to differ-

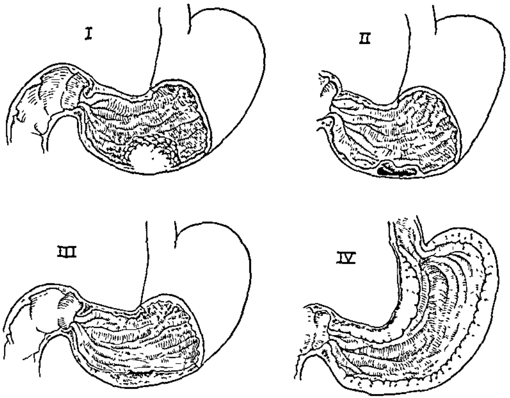

FIG. ur *gross characteristics. I,* exophytic cer. *III,* infiltrative-ulcerative cance . (After Borrmann.)

entiate except by microscopic examination. It is more likely that *gastric ulcerations are either benign or malignant from their inception.*

Gastric Polyps.—Polyps of the stomach are rare compared with polyps of the colon, but gastric polyps are often malignant or are found in association with gastric cancer.

Chronic gastritis.—Some pathologists consider the atrophic form of chronic gastritis to be a precursor of gastric cancer. The issue remains a controversial one.

Pernicious Anemia.—The incidence of gastric cancer is said to be

about three times greater in patients with pernicious anemia than it is in normal patients of comparative age.

Gastric cancer is most common in the lower segment of the stomach, including the prepyloric, antral and lesser curvature areas. Carcinomas of the fundus and greater curvature are less common but often more insidious because they may cause no disturbance in gastric emptying.

Most gastric cancers are adenocarcinomas, which tend to infiltrate the gastric wall, to grow into the gastric lumen and to ulcerate. In addition, they spread beyond the stomach into regional and distant tissues by infiltration and metastasis.

There are four gross types of gastric cancer. Why such wide variations in the morphology of histologically similar tumors occur is unknown; but it is apparent that, once the growth pattern is set, it does not change. Borrmann has proposed the following classification (see Fig. 64):

Type I: polypoid tumors which project into the gastric lumen. The lesion may resemble a mushroom. Ulceration occurs late.

Type II: ulcerated lesions with an elevated margin. There is a sharply demarcated margin and localized infiltration of the gastric wall.

Type III: infiltrative lesions with ulceration. The margins are not sharply demarcated.

Type IV: diffusely infiltrative lesion with ulceration. The tumor may involve a segment of the gastric wall or the entire organ (linitis plastica).

It should be apparent that the clinical signs and symptoms produced by gastric cancer vary greatly according to the location of the tumor, the type of lesion, the degree of ulceration, etc. In general, the prognosis for cure is somewhat better in Types I and II than in Types III and IV, but the appearance of the tumor bears no close relationship to its curability. The prognosis is more dependent upon the extent to which tumor has spread beyond the stomach.

Dissemination of gastric cancer occurs by direct extension to contiguous structures, by lymphatic (Fig. 65) embolization and permeation and by embolization through the venous system. Occasionally, there is direct transperitoneal and gravitational spread.

SYMPTOMS AND SIGNS.—There are no early symptoms of gastric cancer. This disease characteristically has a long silent interval, after which "minor" complaints appear. These include: (1) a tired feeling with loss of "pep"; (2) loss of appetite and loss of weight; (3) mild

age of forty but is not rare in younger people. Most often it develops in the absence of pre-existing gastric disease. However, a few conditions are regarded as precursors of gastric cancer. These include:

Gastric Ulcer.—Probably less than 5 per cent of the gastric ulcers undergo malignant degeneration. Of greater importance is the fact that benign and malignant gastric ulcers are often impossible to differ-

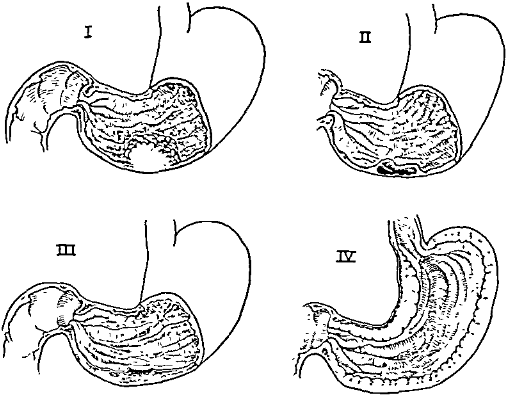

FIG. 64.—Types of gastric cancer according to their gross characteristics. *I*, exophytic polypoid cancer. *II*, ulcerative-infiltrative cancer. *III*, infiltrative-ulcerative cancer. *IV*, diffuse infiltrative cancer (linitis plastica). (After Borrmann.)

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Pernicious Anemia.—The incidence of gastric cancer is said to be

common. Proved dissemination to these areas indicates incurable disease.

The clinical diagnosis is dependent, to a large degree, on x-ray examination. The x-ray criteria of gastric malignancy include: (1) a filling defect, (2) disturbed motility or delayed emptying and (3) distortion of the mucosal pattern.

Gastroscopic examination is sometimes helpful when the x-ray findings are inconclusive. There is, as yet, no widely available practical means whereby a biopsy specimen of the gastric mucosa can be obtained through the gastroscope.

Laboratory studies may help to establish the diagnosis. The finding of blood in the stool and the gastric washings will indicate the presence of ulceration and the need for further investigation. Studies of gastric secretion should be made. Histamine achlorhydria associated with gastric ulceration points strongly to malignant disease, but normal secretion does not rule out or decrease the likelihood of gastric cancer. Hypochlorhydria may have no special significance. It should be kept in mind that many normal individuals have an anacidity to histamine stimulation, the incidence being about 30 per cent in old age.

Cytologic studies of exfoliated cells present in the gastric aspirate are occasionally diagnostic. However, the difficulties associated with obtaining a satisfactory sample and with interpreting the cell changes have limited the usefulness of this technic for the study of gastric lesions.

TREATMENT.—The only cures for gastric cancer have resulted from radical gastric resection before the tumor has extended beyond the operative field. There are no other effective measures. Unfortunately, about half of all the patients with gastric cancer seen by surgeons already have incurable disease. Patients who have proved advanced incurable disease are not subjected to abdominal operation. Other patients are simply explored, a biopsy specimen obtained, and the abdomen closed. A few patients are subjected to palliative resection or short-circuiting. The remaining patients, in whom a more favorable situation is encountered, are subjected to radical operations with some hope of cure.

The operation commonly performed for gastric cancer consists of resection of the distal stomach, gastrohepatic and gastrocolic omenta and regional lymph nodes. Often the spleen and a portion of the pancreas are also removed. The operation is more extensive than, but

indigestion, especially after meals; and (4) a bout of "flu" which does not clear up.

The symptoms associated with cancer of the stomach appear when there is interference with gastric function, outlet obstruction, extensive ulceration, blood loss and anemia, and impaired nutrition. Then pro-

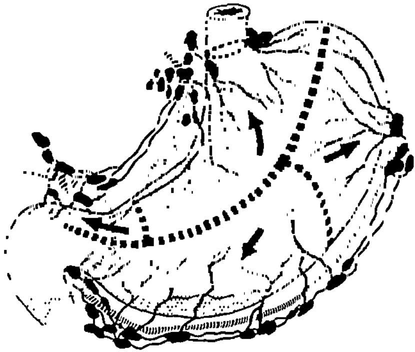

FIG. [illegible]
watershed [illegible]
the stoma[illegible]
area, the porta hepatis, the subpyloric area, the greater curvature region and the gastrosplenic area

tracted pain, vomiting, anorexia, weight loss, pallor and often an abdominal mass appear.

Furthermore, there are no early physical signs of gastric cancer. Not infrequently, marked weight loss, which the patient believes due to "dieting," may be the only indication for further investigation. With progression of the tumor the patient's condition deteriorates and the late signs appear. Sometimes an area of epigastric fulness or a palpable tumor is found. The liver is often enlarged and nodular, and there may be ascites. Sometimes tumor implants are found in the umbilicus or the pelvis (rectal shelf metastases). A spread to the lungs and the left supraclavicular lymph nodes (sentinal or Virchow's node) is

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GASTRIC and ESOPHAGEAL CANCER

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otherwise similar to, that for peptic ulcer. In cases of high-lying gastric cancer, total gastrectomy should be considered.

Palliative surgical measures include: partial gastric resection, gastrojejunostomy to by-pass an obstructed area and jejunostomy for feeding purposes. Palliative procedures in which the tumor is not removed do not prolong the patient's survival significantly. They may, however, give a measure of relief from pain and improved nutrition.

The mortality from gastric resection for cancer ranges between 5 and 10 per cent. The five year survival ("cure" rate) averages about 10 per cent. In round figures, the results of treatment for all patients with this disease may be summarized thus: Out of 100 patients with gastric cancer, about 75 will have exploratory operations; of these, 50 will have a gastric resection with some hope of cure; approximately 5 will die postoperatively; and only 10 will be living and well at the end of five years. The outlook in gastric cancer is indeed grim.

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posed largely of fibrous tissue and the intrinsic vascular and nervous elements of the bowel. (3) The muscular coat outside the submucosa consists of an inner circular layer and an outer longitudinal layer. These last two elements are responsible for muscle tonus and peristalsis. (4) The outer surface of the bowel is covered by the visceral peritoneum.

The blood supply of the midgut is derived from the superior mesenteric artery, the branches of which fan out into the mesentery to form the jejunal and ileal arteries and the ileocolic and right colic arteries. The veins follow the arteries and drain into the portal system. Through this pathway much of the nutriment absorbed in the intestine is carried to the liver.

The intestine is richly supplied with lymphatics. Normally, fat is conveyed in an emulsified state through the lacteals and lymphatics to the thoracic duct and thence to the systemic circulation. The lymphatic vessels of the bowel wall also communicate with the lymph nodes of the mesentery and retroperitoneal area through a rich meshwork of channels.

The nerve supply of the midgut segment is derived from the autonomic nervous system. Of some importance to the clinician is the fact that visceral pain arising from any segment of the midgut is characteristically referred to the midzone of the abdomen through autonomic communications in the cord (T9 to L1).

Large volumes of fluid enter the jejunum from the stomach and duodenum. The amount of digestive secretion alone is 8–10 L. a day. Here the food and digestive secretions are mixed, digestion begins, and the osmotic tension of the mixture is balanced with that of the blood by the addition of jejunal secretion. As the mixture moves into the ileum, foodstuffs are absorbed. When alterations in conduction, secretion or absorption occur, the balanced activity of the intestine is upset and nutrition is impaired.

Important disorders of the midportion of the alimentary canal include: midgut volvulus and Meckel's diverticulitis, intestinal obstruction, regional ileitis, tumors of the small intestine, acute appendicitis and cancer of the cecum and the right side of the colon. Midgut volvulus and Meckel's diverticulitis are considered in Chapter 24, on Abdominal Conditions of Infants and Children. Cancer of the cecum and right colon is discussed in Chapter 16, on The Lower Alimentary Canal.

CHAPTER 15

The Midalimentary Canal

THE PORTION of the alimentary canal that extends from the duodenal papilla to the midtransverse colon is derived from the midgut embryologically. It is concerned with the conduction, dilution, digestion and absorption of foodstuffs. It includes the second, third and fourth portions of the duodenum, the jejunum, the ileum and the right half of the colon. The union between the small intestine and the colon is marked by the ileocecal valve, which controls the entrance of material from the small bowel into the cecum and normally prevents reflux into the small bowel.

The duodenum and the jejunum are continuous at the duodenojejunal flexure. The duodenum measures about 25 cm. in length and is covered by peritoneum on its anterior aspect only. The jejunum (upper two fifths) and ileum (lower three fifths), which constitute the remaining small intestine, are of variable length but average about 7 m. (23 ft). The juncture of the jejunal and ileal segments is not distinguished by any definite anatomic structure; but the jejunum is regularly thicker walled, larger in diameter and more richly supplied with blood vessels. The jejunum and ileum are suspended by, and receive their blood, lymphatic and nerve supply through, the small-bowel mesentery. The base or root of the mesentery arises along an oblique line, 20 cm. in length, extending retroperitoneally from the left side of the second lumbar vertebra to the ileocecal junction on the right.

The intestinal wall consists of four layers. (1) The mucosa forms a continuous inner lining, the absorptive surface of which is increased by the presence of circular folds (plica circulares) and innumerable villi. (2) The submucosa, immediately beneath the mucosa, is com-

posed largely of fibrous tissue and the intrinsic vascular and nervous elements of the bowel. (3) The muscular coat outside the submucosa consists of an inner circular layer and an outer longitudinal layer. These last two elements are responsible for muscle tonus and peristalsis. (4) The outer surface of the bowel is covered by the visceral peritoneum.

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INTESTINAL OBSTRUCTION

As Coller said*:

> If there is mechanical obstruction of the small bowel, the ideal time to operate on the patient is before dehydration, before distention and before infection occur. . . . Even though decompression is accomplished, it is much better to carry out operative correction of the mechanical defect as soon as the patient is in condition to withstand operation. When one is in doubt concerning the patient seen early with generalized colicky pain, nausea and vomiting, even though he cannot make out the exact location of the obstruction he should advise operation.

General Considerations

Any condition which interferes with the normal forward motion of bowel content results in intestinal obstruction. The obstruction may be located either in the small or the large intestine; it may be incomplete or complete; it may be chronic, subacute or acute; it may be simple or complicated (e.g., strangulating, "closed loop"); or it may be recurrent or established.

Intestinal obstruction is usually the result of one of three primary disturbances: (1) mechanical blockage of the lumen, (2) reduced or absent peristalsis due to neurogenic factors (paralytic or inhibition ileus) or (3) impaired circulation due to vascular occlusion.

Mechanical occlusion of the bowel is the most common cause of intestinal obstruction. It results from abnormalities located within the bowel lumen (intraluminal), within the bowel wall (intramural) or outside the bowel wall (extraluminal). Once obstruction becomes established, a variable degree of interference with the neurogenic and vascular elements of the bowel also develops. That is, in the later stages of acute mechanical obstruction with distention, peristalsis is reduced and blood flow to the bowel wall is impaired. Thus, elements of both neurogenic and vascular origin are superimposed.

Reduced or absent peristalsis results from functional impairment of the intrinsic or extrinsic autonomic nervous mechanisms. Some degree of ileus occurs after abdominal operations from local injury to the bowel or peritoneum, as well as from peritonitis from any cause, and also as a part of the physiologic response to any severe injury. The reduction in peristalsis may be physiologic or pathologic,

*Coller, F. A., and Buxton, R. W.: Acute obstruction of the small bowel, J.A.M.A. 140:135, 1949.

according to the severity of the local or systemic insult to the bowel.

Circulatory impairment leads to ischemia and a reduction in all bowel functions. The circulation may be cut off beyond the bowel wall (extramural strangulation) from mesenteric thrombosis or embolism, or from torsion of the mesentery (volvulus), or within the bowel wall (intramural strangulation) as a result of prolonged distention. The ischemic segment represents an area of obstruction despite the fact that the lumen is open, because bowel content cannot be propelled distally.

High intestinal obstructions generally cause more serious disturbance of bodily functions than low obstructions. The clinical picture is more acute, and deterioration is more rapid. This difference relates to the relatively greater loss of electrolyte-rich fluid both into the bowel and externally (vomiting) and to the unavailability of the great absorptive surface below the obstruction. Unless the obstruction is relieved, dehydration, acid-base imbalance (usually acidosis), hemoconcentration, shock and death occur.

Distention develops above the obstruction as a result of stasis of bowel content. The material in the bowel consists of ingested food and fluid, digestive secretions and gas. Interference with the normal secretion-absorption functions of the mucous membrane results in an accumulation of abnormal quantities of digestive secretions. Abnormal quantities of gas also appear above the obstruction. The main source of gas, about 70 per cent, is swallowed air; about 20 per cent of the gas is derived from the blood by diffusion; and the remainder, 10 per cent, results from bacterial fermentation.

Distention of the intestine is the most important single pathophysiologic consequence of obstruction (Fig. 66), and it is often the most difficult to control. The appearance of distention is followed by a cycle of events which, if not interrupted, leads to death. Severe intestinal distention is a self-perpetuating mechanism, causing progressive distention. The passage of fluid and gas is blocked, and the absorption of fluid and gas is impaired by virtue of the distention. Secretion from the bowel wall is augmented and motility is inhibited by the same mechanism. The blood, lymphatic and tissue fluid interchanged in the bowel wall is reduced by intramural vascular compression, angulation and edema. The distention leads to: (1) metabolic derangements (fluid, electrolyte, decreased blood volume, shock, etc.); (2) respiratory and cardiovascular derangements from increased intra-abdominal pressure; and (3) local changes in the bowel

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Mechanical obstructions usually require surgical treatment; neurogenic obstructions, nonsurgical treatment; and obstructions resulting from vascular impairment, excision of the damaged bowel. In all types of intestinal obstruction, it is essential that the metabolic disturbances be corrected promptly and that the adverse effects of distention on ventilation and circulation be minimized.

Clinical Considerations

The treatment of early simple intestinal obstruction should not be difficult and the mortality should be low; the treatment of late cases is often complicated and the mortality is high. The key to successful management lies in early diagnosis and treatment. Intestinal obstructions are generally emergencies.

Intestinal obstruction should be considered in every patient in whom abdominal pain is the chief complaint. Often this diagnosis can be ruled out or established without too much difficulty. Errors in diagnosis result when the possibility of intestinal obstruction is overlooked or when complications of obstruction, which are usually the result of delay, cloud the clinical picture.

The first question that must always be considered—and answered—is: "Does the patient have intestinal obstruction?" Other questions may or may not be answered immediately, but failure to answer these does not preclude operation. Such questions are: "What is the cause of the obstruction?" "Where is the obstruction?" "Is the obstruction complete or incomplete?" "Is it simple or strangulating?"

To further clarify this point, it can be said that operation for intestinal obstruction may properly be undertaken without a precise knowledge of its cause, location, completeness and complications. To some extent, these factors may be predicted from the clinical findings, but definite answers to the problem often depends on abdominal exploration. In this regard, the more one attempts to establish a precise diagnosis by delaying definitive treatment, the more likely it is that the patient will be harmed.

Symptoms and Signs

Abdominal pain is the outstanding symptom. It is characteristically severe, poorly localized, colicky and intermittent. The pain may be slight at onset but becomes more severe as time passes.

wall from ischemia, including edema, ulceration of the mucous membrane, necrosis of the bowel wall, and infection, perforation and peritonitis.

Much has been written about a "toxic" factor in advanced intestinal obstruction. Presumably there exist certain products of bacterial growth or tissue breakdown which enter the circulation

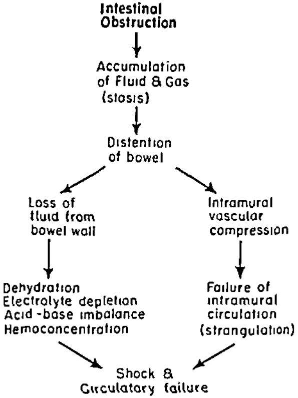

FIG. 66.—Pathophysiology of intestinal obstruction.

through the bowel or through peritoneal absorption when the bowel wall becomes nonviable. The exact nature of these substances is unknown, but certain enzymes and hemoglobin derivatives have been incriminated. It has been demonstrated that the onset of the "irreversible" shock of advanced intestinal obstruction can be delayed by the administration of broad-spectrum antibiotics, either locally into the bowel or systemically.

This vicious cycle of events which follows obstruction can be prevented by removing the obstruction and by preventing or relieving distention, or by the combined relief of obstruction and distention.

bladder, the urinary tract and the female pelvic organs may resemble intestinal obstruction. In addition, perforated peptic ulcer, which is common, and acute pancreatitis, which is uncommon but easily missed, should also be considered. These conditions are considered in Chapter 21, on Acute Surgical Conditions of the Abdomen.

Causes of Mechanical Obstruction

Occlusion of the intestinal lumen may result from something which blocks the lumen from within, from something which narrows the bowel wall or from something which compresses the bowel from without. There are obviously innumerable causes of intestinal obstruction, and it hardly seems necessary for the student to memorize such a list. Instead, if the student considers the location of obstructions with respect to the bowel wall, and the origin of various conditions which cause obstruction, he will be able to devise a list. The usual bases for all pathologic processes are: congenital and acquired defects, inflammatory and infectious conditions, benign and malignant diseases and, in the case of hollow organs, obturation or blockage from within, as from a foreign body.

The majority of acute intestinal obstructions in the adult are the result of strangulated hernia, intraperitoneal adhesions and neoplastic diseases inside or outside the bowel (primary or secondary). Obstructions in infancy are due to developmental defects, such as stenosis or atresia, malrotations, congenital megacolon and imperforate anus. In childhood, obstructions may be the result of intussusception, volvulus, Meckel's diverticulum, appendicitis, etc.

The most frequent causes of intestinal obstruction are:

Mechanical Occlusion

	Adults	*Infants and Children*
Most common:		
	Strangulated hernia	Congenital atresia and stenosis
	Peritoneal adhesions	Imperforate anus
	Tumors, benign and malignant, of bowel and other abdominal organs	Malrotations
		Intussusception, usually ileocolic
		Megacolon (Hirschsprung's disease)
Less common:		
	Volvulus, usually sigmoid	
	Gallstone obturation	
	Intraperitoneal abscesses	
	Fecal impaction	

Vomiting occurs shortly after the pain in high obstructions, usually later in low obstructions. It is often absent in large-bowel obstructions. Initially the vomitus consists of stomach contents and bile; later it may become fecal-like in appearance and odor.

Distention of the bowel causes distention of the abdomen. In high obstructions there may be distention of only a short segment of bowel and no apparent abdominal distention. In low obstructions some degree of abdominal distention is regularly found.

Obstipation or *constipation* are symptomatic. One or more bowel movements may follow the onset of obstruction; obstipation follows if the obstruction is complete. Constipation is usual with incomplete obstruction, but the absence of this symptom does not exclude this diagnosis.

Hyperactive peristalsis is also a symptom. In mechanical obstructions there is exaggerated bowel activity above the obstruction. The abdomen becomes noisy and borborygmi are heard. On auscultation, there will be peristaltic rushes and rumbles, often with a metallic tinkling quality, which are synchronous with colic. If looked for, visible peristalsis is often observed.

X-ray scout films are often diagnostic. The bowel loops above the block are distended with gas and fluid. In the upright film, "fluid levels" are found. The small bowel may be arranged in a stepladder fashion. Usually the folds of mucous membrane of the upper small bowel (plica circulares) are prominent. The colon is distinguished by its haustral markings and the distribution of the distended bowel.

The *vital signs* (temperature, pulse, respiration) are not significantly altered in the early stages of simple intestinal obstruction. In advanced obstruction with local and systemic changes, alterations occur. There is slight fever and tachycardia. The respiratory rate rises by virtue of impaired ventilation or acidosis, or both. The urine becomes concentrated, is acid in reaction and contains ketone bodies as a result of starvation. Hemoconcentration occurs and leukocytosis develops.

Differential Diagnosis

Mechanical obstructions of the small intestine often simulate other acute conditions of the abdomen. The conditions most likely to be confused are those in which pain (colic) of smooth-muscle origin is the outstanding symptom. Thus, diseases of the appendix, the gall-

pected complication of acute intestinal obstruction. Directly or indirectly, it is the cause of most deaths from obstruction. It can be produced by incarceration of the bowel in the neck of a hernial sac, by twisting of the bowel upon its mesentery (volvulus) or by fixation of a loop by an adhesive band. The mesenteric circulation is blocked, and tissue necrosis follows. The necrotic area is penetrated by bacteria, and localized or diffuse peritonitis develops. The necrotic segment becomes purple or black and may rupture if the patient lives long enough.

Possible strangulation of the bowel should be considered in every patient with acute intestinal obstruction, and special weight should be given to this possibility if there is long-standing obstruction. The signs which point to strangulation are: (1) sudden, severe and continuous abdominal pain; (2) severe localized abdominal tenderness, often with a palpable sausage-like loop of bowel; (3) muscle spasm or other evidence of peritoneal irritation, (4) absence of peristalsis on auscultation; (5) a severe systemic reaction, often including shock out of proportion to what is usually encountered; and (6) a poor response to supportive treatment.

Treatment

Management of the patient with intestinal obstruction requires careful evaluation of the individual problem with respect to the local and systemic changes, good clinical judgment, good timing and skilful application of surgical and supportive measures.

The objectives of treatment are: (1) to relieve the obstruction at the earliest time, consistent with safety to the patient; (2) to relieve distention of the bowel as completely and rapidly as possible; (3) to restore fluid, electrolyte and hemodynamic balance; and (4) to prevent or treat infection

A short period of preoperative preparation is usually necessary. The upper gastrointestinal tract should be emptied by means of nasogastric suction. Intravenous electrolyte-containing fluids (including potassium) should be administered. The bladder should be emptied. Morphine or related drugs should not be administered until the diagnosis has been established and treatment has been outlined. The necessary x-rays (usually flat and upright films of the abdomen) can be obtained before or after institution of the foregoing measures, depending on the patient's condition.

REDUCED PERISTALSIS (paralytic or inhibition ileus)

Operative trauma
Peritonitis
Severe injuries, all types
Severe infections, all types

IMPAIRED CIRCULATION (strangulation of vascular supply)

Mesenteric artery or vein thrombosis
Mesenteric artery embolism

The clinical history, physical findings and x-ray studies may suggest the cause and location of the obstruction. For example: Obstructions in the neonatal period are almost all due to errors in development. Obstructions appearing before the age of two years are commonly the result of intussusception, the clinical picture of which is quite characteristic. Obstructions caused by strangulation in a hernia are usually apparent. Obstructions which result from intraperitoneal bands are almost always associated with an old or new operative scar. Obstructions due to tumors are likely to be associated with evidence of chronic illness if malignant, or a palpable mass or known pelvic disease. Gallstone obturation, which is uncommon and often missed, follows an acute exacerbation of gallbladder disease.

The general location of the obstruction may be suggested by the severity of the symptoms, which tend to be inversely related to the length of the obstructed segment. The degree of abdominal distention bears a direct relationship to the length of distended bowel and may be correlated with the distribution, number and appearance of dilated loops seen on the x-ray films. From these observations it is usually possible to conclude that the obstruction lies either high or low in the small bowel or in the colon. Barium studies of the colon are helpful if large-bowel obstruction is suspected. *Barium should not be given by mouth to patients with acute intestinal obstruction.*

The question as to whether the obstruction is complete or incomplete is frequently not answerable immediately and may, in fact, be of academic interest only. Under conditions of established intestinal obstruction the treatment is surgical, regardless of the completeness of the block. When the clinical picture is one of intermittent obstruction, with borderline signs and without cessation of gas and feces by way of the rectum, there is a place for nonoperative treatment by tube decompression. This condition is often encountered in patients with recurrent obstructions due to intraperitoneal adhesions.

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pected complication of acute intestinal obstruction. Directly or indirectly, it is the cause of most deaths from obstruction. It can be produced by incarceration of the bowel in the neck of a hernial sac, by twisting of the bowel upon its mesentery (volvulus) or by fixation of a loop by an adhesive band. The mesenteric circulation is blocked, and tissue necrosis follows. The necrotic area is penetrated by bacteria, and localized or diffuse peritonitis develops. The necrotic segment becomes purple or black and may rupture if the patient lives long enough.

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The type of operation required will depend on the nature of the obstruction. The operation should be as direct and atraumatic as possible, yet extensive enough to relieve the obstruction. In obstruction due to a single adhesion (Fig. 67), the operation may be simple; if strangulation is encountered, the operation is more difficult. All necrotic bowel must be removed and bowel continuity re-established. Deperitonealized areas should be covered over, if possible.

The operation can be performed under either spinal or general

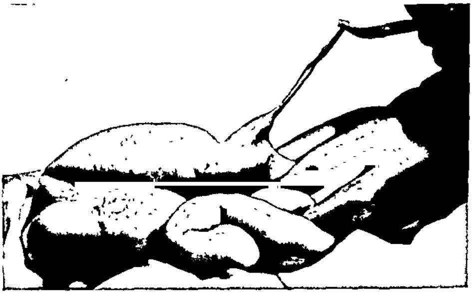

FIG. 67.—Acute intestinal obstruction due to peritoneal adhesion or band (photograph taken at operation). Note the long fibrous band which fixed and angulated the bowel at the site of previous hysterectomy. The bowel above the point of obstruction is *markedly distended with fluid and gas; the bowel below is of normal size.*

anesthesia. The point of obstruction is located by identifying the distal collapsed small bowel and following it upward. Distended bowel must be handled with great care because it is torn easily, in which case peritoneal soiling occurs. Nonviable bowel is recognized by its color (purple or black), absence of peristalsis, failure to gain its normal color after release of circulation and lack of pulsations in the arteries.

Suction-decompression is a useful method for treating intestinal distention. It may be the sole measure required for patients with partial or intermittent obstruction. It is an important adjunctive measure in the treatment of all patients before and after operation.

Suction-decompression is accomplished by the introduction of a rubber or plastic nasogastric tube (Fig. 68, *A*) or long intestinal tube (Fig. 68, *B*) to which a continuous source of negative pressure is applied. The Wangensteen suction device is commonly used. Such a system removes fluid and gas above the obstruction and decreases or prevents distention. Because much of the fluid and salt removed is important to body economy, these substances must be replaced through parenteral routes.

The *long intestinal tube* is designed to enter and effect decom-

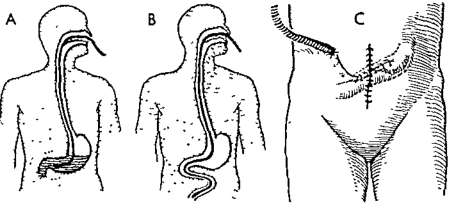

FIG. 68.—Technics of decompression of upper gastrointestinal tract. *A*, nasogastric tube aspiration *B*, long intestinal tube aspiration. *C*, operative enterostomy (a Witzel type tube enterostomy is illustrated, although this procedure is infrequently used today).

pression of the small bowel. Several types of long tubes are available. Probably the most widely used is the single-lumened Cantor tube. Attached to its tip is a rubber bag containing mercury, which aids passage of the tube into the duodenum and small bowel. The tube is introduced through the nose into the stomach and is then carried by peristalsis into the small bowel. Passage may be delayed or prevented at the pyloric barrier, usually, however, the tube enters the duodenum within four to six hours and progresses downward as decompression occurs. This device must be used with greatest care. It is not a substitute for operation, nor should it be used to delay an indicated operation. In inexperienced hands it may do more harm than good

Operative enterostomy (Fig. 68, *C*) is a third form of decompression technic now rarely used.

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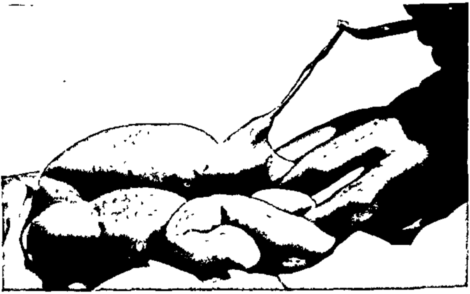

FIG. 67.—Acute intestinal obstruction due to peritoneal adhesion or band (photograph taken at operation). Note the long fibrous band which fixed and angulated the bowel at the site of previous hysterectomy. The bowel above the point of obstruction is markedly distended with fluid and gas, the bowel below is of normal size.

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possible and operation becomes necessary to establish the diagnosis.

Chronic Diarrhea Resembling Ulcerative Colitis.—The symptoms are those of chronic enteritis, with anemia, weight loss, recurrent attacks of abdominal pain, nausea, vomiting and diarrhea.

Chronic Intestinal Obstruction.—Edema and fibrosis produce narrowing of the intestinal lumen; and the narrowing is often enhanced by angulation, fixation and fistula formation. This leads to mechanical blockage of varying degree and signs of intermittent or partial intestinal obstruction.

Bowel Fistulas.—Fistulas result from ulceration, perforation, abscess formation and erosion of contiguous structures. Fistulous tracts may be internal (communicating with another hollow organ) or external, to the abdominal wall. A persistent abdominal fistula following appendectomy suggests the possibility of regional ileitis.

Differential Diagnosis

During the acute phase of regional ileitis it may be impossible to exclude acute appendicitis without operation. In the chronic stage, the diagnosis is often suspected from a history of repeated bouts of abdominal pain, disturbed bowel function, a palpable thickened loop of bowel or an abdominal fistula. Regional ileitis may masquerade as "chronic appendicitis," ulcerative colitis, diverticulitis, amebic dysentery or carcinoma. Diagnosis is established by x-ray studies of the small bowel (progress meal). The "string sign" is indicative but not pathognomonic of regional ileitis. This finding is represented by a narrowed segment of bowel which contains a thin stream of barium, resembling a piece of string.

Treatment

There is no well-established form of treatment for regional ileitis. Failures occur with both medical and surgical measures. There is much to be said in favor of nonoperative treatment when complications do not pose a problem. In some instances of acute regional ileitis, spontaneous healing probably occurs. In the chronic stages, resection of the diseased segment or segments often becomes necessary. Because the possibility of recurrence and the need for subsequent resections always exists, there is danger that radical treatment may ultimately leave the patient with insufficient small bowel to

REGIONAL ILEITIS

Crohn and his asociates first described the condition as a separate disease entity (1932) in the following words: "A disease of the terminal portion of the ileum, affecting mainly young adults, characterized by disproportionate connective tissue reaction in the remaining walls of the involved intestine, a process which frequently leads to stenosis of the lumen associated with the formation of multiple fistulae." * Actually, little has been added to the knowledge of this disease since Crohn's original studies. The cause of the disease is unknown, but it has many features which suggest an infectious etiology.

Although the terminal ileum is most commonly involved, any portion of the small bowel, and in some instances portions of the large bowel, may be involved. Sometimes there is more than one diseased segment, with intervening normal or "skip" areas. The disease tends to be chronic with exacerbations and partial remissions. As the disease progresses, complications develop which require surgical treatment.

The distinguishing pathologic characteristics of regional enteritis are as follows: (1) In the early stages the serosa is inflamed and the bowel wall is edematous. (2) Later there is thickening and hypertrophy and the bowel wall becomes rigid and fibrotic. The lumen is also narrowed. (3) Ulceration of the mucosa and perforation are followed by the formation of fistulas between the bowel and surrounding structures. (4) The mesentery is thickened, edematous and contains many large, soft lymph nodes.

Clinical Picture

The disease is usually encountered in patients twenty to forty years of age. The following four patterns of regional ileitis are recognized:

Acute Abdominal Complaints Resembling Appendicitis.—Pain may be localized to the right lower quadrant, but it is more often diffuse Vomiting and bowel disturbances are common. Abdominal tenderness and muscle spasm appear, depending on the extent of involvement. Fever and leukocytosis may be expected. The disease may mimic acute appendicitis so closely that differentiation becomes im-

* Crohn, B. B., Ginzburg, L., and Oppenheimer, G. D.: Regional ileitis: A pathologic and clinical entity, J.A.M.A 99:1323, 1932.

skin with cardiac failure, due to excessive circulating serotonin produced by hepatic metastases from carcinoid tumors of the small bowel or colon.

APPENDICITIS

Acute appendicitis is the most common acute surgical condition of the abdomen, accounting for about 60 per cent of all emergency laparotomies. It occurs in both sexes and at all ages, but with greatest frequency in the second and third decades of life. The clinical picture may be clear-cut and diagnosis may be simple; but oftentimes, and especially in the very young and the very old, recognition is difficult. Diagnosis may be difficult because the history is incomplete or unobtainable, because the symptoms and signs are "atypical," because the local and systemic reactions are so severe or so mild that the true origin of the disease is obscured or because delay and misdirected treatment have fogged the picture so that the complications of appendicitis, rather than the primary disease, is treated.

In the past, cathartics have commonly been given for acute abdominal pain. Fortunately, this practice is decreasing. The dangers inherent in a policy of cathartics and delay in appendicitis have been emphasized and re-emphasized to the public and the profession alike. The knowledge of these dangers, together with the wide availability of trained physicians who understand the urgent nature of appendicitis as well as the effectiveness of early surgical treatment, has resulted in a striking decrease in the mortality from this disease and its complications.

Appendicitis usually appears suddenly in an otherwise healthy person. In about 80 per cent of instances an obstruction to the appendical lumen can be shown to be the initiating cause. The obstruction may be due to a fecal concretion (fecalith), an adhesive band or kink, parasites such as pinworms, submucosal lymphoid hyperplasia or a tumor of the appendix or the cecum. When appendicitis follows in the wake of an acute infection such as a "cold," the "flu," tonsillitis or an exanthem, it is likely that a diffuse reaction of the reticuloendothelial system, including these elements of the appendix, sets the stage for appendical obstruction and infection.

When the appendical lumen becomes blocked, a "closed loop" obstruction is produced and a vicious cycle of events is initiated (Fig. 70), which, unless relieved, leads to appendical necrosis and peri-

maintain health. As an alternative, the diseased bowel may simply be by-passed and left in situ, by establishing an enterocolostomy. This is a more conservative approach and is advocated by many surgeons.

TUMORS OF THE SMALL BOWEL

Primary tumors of the small intestine are rare. The most frequent benign tumors are the adenomas, fibromas, leiomyomas and lipomas. The most frequent malignant tumors are the adenocarcinomas, sarcomas, lymphomas and carcinoids.

There are no constant early symptoms of intestinal tumors. Ulcer-

FIG. 69.—Spotty pigmentation of the lower lip, as well as of the oral mucous membrane, in a 9 year old child with multiple polyps of the small bowel (Peutz-Jegher's syndrome)

ation and hemorrhage may result in unexplained anemia. Intermittent signs of intestinal obstruction occur as the tumor enlarges or as it contracts and blocks the lumen Pedunculated tumors may produce intussusception The diagnosis of tumor should be suspected if other causes for disturbed bowel functon have been excluded X-ray studies may support the diagnosis, but final diagnosis usually requires abdominal exploration.

Two rare intestinal tumors which are associated with interesting clinical findings should be mentioned. The first is the genetic-linked syndrome of intestinal polyposis and spotty (melanin) pigmentation of the lips and oral mucous membrane (Peutz-Jegher's syndrome, Fig. 69). The second is the syndrome of intermittent flushing of the

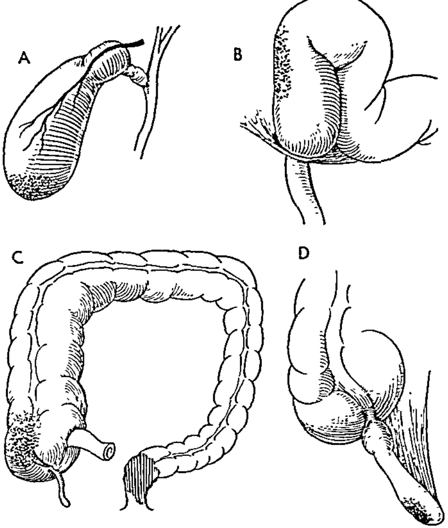

Fig. 71.—Common sites of tension gangrene of hollow organs, produced by obstruction, distention, ischemia and infection. *A*, acute obstructive cholecystitis due to a stone impacted in the neck of the gallbladder. *B*, acute small-bowel obstruction due to peritoneal adhesions. *C*, acute large-bowel obstruction due to cancer of the sigmoid with competent ileocecal valve ("closed loop" obstruction). *D*, acute obstructive appendicitis due to fecal concretion. (After Saint, J. H.: Acute cholecystitis and its rational treatment, Surg., Gynec. & Obst. 75:323, 1942.)

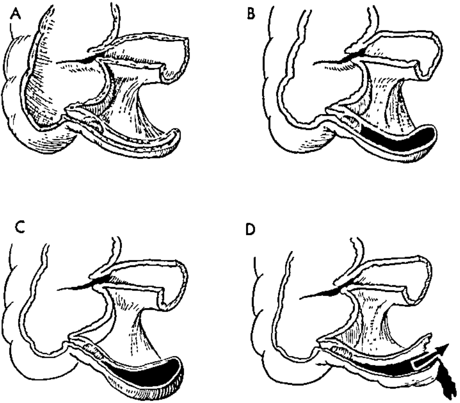

FIG 70.—Pathogenesis of acute obstructive appendicitis with rupture and peritonitis. *A*, fecalith in the appendical lumen. *B*, obstruction to lumen ("closed loop" type) and stasis of fecal material containing virulent pathogenic organisms. *C*, mucus, exudate and products of bacterial growth collect and produce inflammation. *D*, intraluminal tension mounts, intramural vascular strangulation occurs and gangrene with perforation of the appendical wall follows. Peritonitis ensues.

tonitis. The completeness of the obstruction and the severity of the infection, rather than its duration, will determine the gravity of the process.

The sequence of events in appendical obstruction may be tabulated thus:

Appendical obstruction
Intraluminal distention
Increased secretory activity (of the mucous membrane)
Interference with the venous, lymphatic and arterial flow
Impaired viability and increased permeability of the appendical wall
Bacterial growth and invasion
Necrosis and tension gangrene (Fig. 71)
Perforation, usually antimesenteric in location
Peritonitis, local or diffuse

and peritonitis in a matter of eight to twelve hours. In others, it is *mild, remitting* or "grumbling" and persistent over a period of several days. Not infrequently, appendicitis clears spontaneously, but this is not always predictable. Between these two extremes are many patients with appendicitis who exhibit more or less the classic pattern, to be described below.

A history of previous attacks of abdominal pain suggestive of appendicitis is often elicited and may serve to reinforce the diagnosis. One must keep in mind that the course of appendicitis may be modified by treatment with antibiotics, cathartics and possibly enemas. Analgesics, especially morphine, will also cloud the physical signs and make assessment more difficult.

Clinical Considerations

It is well to consider any acute abdominal pain which persists for *six hours or more* as due to appendicitis until proved otherwise. Commonly the symptoms follow a definite sequence. First there is periumbilical or epigastric pain, which may be intermittent and colicky in nature This is followed by nausea and often vomiting. The patient may vomit once or twice, but vomiting is generally not severe or protracted. Within a few hours the patient complains of pain in the right lower quadrant and there is tenderness on pressure over this region. As the inflammatory process develops, a moderate systemic reaction, manifested by a slight rise in body temperature and elevation in the polymorphonuclear cells in the blood, appears.

The physical signs will depend on the stage of the disease. *Early in the course of appendicitis there may be few, if any, signs*; but soon there will be localized right lower quadrant tenderness, muscle guarding or rebound tenderness. Occasionally, *fulness of the cecum* or an ill-defined mass may be detected. The mass is usually formed by the inflamed appendix covered by the greater omentum. As the local reaction progresses over a period of hours or days, the mass may become more definite, tenderness and muscle spasm may increase, the bowel sounds disappear, and the systemic reaction becomes more severe.

In advanced appendicitis with extra-appendical extension, the patient will exhibit signs of local or diffuse peritonitis, including *rigidity of the abdominal muscles, distention, absence of bowel sounds, protracted vomiting, fever and dehydration.*

The resident bacteria of the cecum and appendix are responsible for the infection. The main pathogens are streptococci (enterococci), staphylococci, coliform organisms and gas-producing anaerobes. The local defense mechanisms may confine the infection to the vicinity of the appendix itself, with the formation of a periappendical abscess; or the protective reaction may be too little and too late, in which case diffuse peritonitis results.

The base of the appendix arises (Fig. 72) from the medial aspect of the cecum at the point of convergence of the three longitudinal

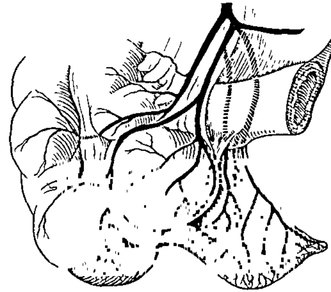

FIG. 72.—Relationship of terminal ileum, cecum and appendix. The ileocolic artery, a branch of the superior mesenteric artery, is the source of blood to this area. Note that the appendix is continuous with the anterior taenia coli.

muscle bands of the colon (taenia coli). The tip of the appendix may lie in any position with respect to the cecum, anterior abdominal wall, retroperitoneal coverings, right upper quadrant or pelvis, depending on the length of the appendix. It is of some help, clinically, to think of this area in terms of three dimensions in order to understand the possible locations of the appendix and how position may influence the signs and symptoms in appendicitis. Suffice it to say that innumerable variations are conceivable and that the findings at operation can often be correlated with the clinical picture.

The course of appendicitis varies from patient to patient. In some patients, the course is rapid and fulminant, leading to rupture

the process may have progressed past the stage of appendical obstruction or that there may be some other cause for the abdominal pain. High fevers are unusual early in the course of acute appendicitis except in children. A history of a chill is against the diagnosis of uncomplicated appendicitis. However, invasive infection of the portal vein (pylephlebitis), which is a rare complication of appendicitis, may be heralded by fever and chills.

7. The total white blood cell count is variable, but averages about 12,000. There is commonly an increase in the polymorphonuclear cells, ranging from 70 to 90 per cent. In advanced appendicitis there is a moderate to marked increase in these values. It must be recalled that the blood findings are nonspecific and must always be interpreted with respect to the total clinical picture.

8. The urine is normal except when the inflamed appendix lies near the ureter or bladder. Under these conditions, pus cells and red blood cells may appear in the urine.

9. X-ray studies are of differential diagnostic value only. Usually they are not needed. When acute intestinal obstruction, acute cholecystitis, acute perforated peptic ulcer or ureteral colic are under consideration, scout films of the abdomen should be obtained.

10. A short period of observation with periodic examination and/or consultation may be required when the diagnosis is in doubt. Often, the issue can be settled only at operation.

Differential Diagnosis

Acute appendicitis must be differentiated from a number of medical and surgical conditions. Many of these conditions are to be described in Chapter 21, on Acute Surgical Conditions of the Abdomen. It seems desirable, however, to review briefly some of the considerations which pertain to acute appendicitis.

Although acute pulmonary and cardiac conditions may mimic appendicitis, they can usually be distinguished by the careful physician. It should be remembered that appendicitis and cardiac or pulmonary disease (or any other condition) may be coexistent. Acute coronary thrombosis and acute lobar pneumonia (right lower lobe) must be considered in all patients with abdominal pain. The history, physical findings and, if indicated, x-ray and electrocardiographic studies will usually establish the diagnosis. An unnecessary operative procedure carried out in the face of an unrecognized coronary throm-

Several diagnostic points need emphasis:

1. The initial pain in appendicitis is usually located in the midabdominal zone, is ill-defined and tends to be colicky. The pain is similar to visceral pain of midgut origin from any cause, whether due to "green apple colic," small-bowel obstruction or acute regional enteritis. The pain and tenderness in the right lower quadrant which appears later is the result of peritoneal irritation from inflammation. The midabdominal pain usually disappears when localization in the right lower quadrant occurs.

2. The subsequent pain and tenderness is referred to McBurney's point. The patient will often indicate this region in response to the request to "point with one finger to where the pain is greatest." McBurney's point is located one-third the distance from the right anterosuperior iliac spine on a line drawn from the iliac spine to the umbilicus.

If one appreciates the numerous variations in the normal position of the appendix, he will understand that McBurney's point will not always be the site of maximum pain and tenderness.

3. Involuntary muscle spasm (*rigidity*) of the abdominal muscles is a protective reflex incited by parietal peritoneal irritation. The degree of muscle spasm can be gauged by comparing the muscle tone on the right side of the abdomen with that on the left. Spasm in other muscles is also observed. The inflamed appendix may lie near the right iliopsoas or the right obturator internus. Spasm of the psoas is indicated by pain and decreased motion when the thigh is extended. Spasm of the obturator is indicated by pain and restricted internal rotation of the thigh when it is flexed on the abdomen. When muscle spasm exists, the patient will usually stoop slightly, walk with a limp and lie with the right thigh flexed.

4. Rebound tenderness is also a sign of peritoneal irritation. In the absence of abdominal muscle spasm, it indicates deep or visceral peritoneal involvement. It is elicited by applying gentle, steady pressure locally, then releasing the pressure suddenly. These manipulations are painful and should be done with the least possible discomfort to the patient. They should not be repeated except for good reason.

5. Rectal tenderness high on the right is common. When the appendix lies in the pelvis, a mass may be palpable. Rectal examination should be a routine part of the physical examination of all patients complaining of abdominal pain.

6. When the body temperature exceeds 102° F., consider that

may be necessary to establish the diagnosis of ureteral obstruction.

Certain acute conditions of the female pelvic organs are often most difficult to differentiate from acute appendicitis. This is true especially when the appendix lies in close proximity to these structures. Most of the difficulties concern acute salpingitis, rupture of an ovarian follicle, ruptured ectopic pregnancy and ovarian cyst with a twisted pedicle. The gynecologic history and examination will usually indicate the pelvic organs to be the site of an inflammatory process. In tubal pregnancy with rupture, a missed menstrual period, a history of vaginal bleeding and signs of internal hemorrhage together with signs of early pregnancy suggest the correct diagnosis. In these conditions, operative treatment will usually be indicated.

From the standpoint of differential diagnosis, the conditions which simulate appendicitis may be classified into three groups, according to the role of operative treatment in their management:

I. *Operation Contraindicated*
   Lobar pneumonia; coronary thrombosis; acute pancreatitis

II. *Operation Contraindicated but Not Necessarily Harmful*
   Bc [illegible]

   Ur [illegible]

   FEMALE PELVIC ORGANS: Ruptured ovarian follicle; pelvic inflammatory disease

III. *Operation Indicated*
   Intestinal obstruction, perforated peptic ulcer; acute cholecystitis (some cases); ruptured ectopic pregnancy, torsion of pedicle of ovarian tumor or cyst; Meckel's diverticulitis

TREATMENT

Appendectomy is indicated for all cases in which the diagnosis of uncomplicated acute appendicitis is made. The operation should be performed as soon as the patient is ready for it, that is, as soon as the essential preoperative care has been completed. There is nothing to be gained by prolonged observation, antibiotic treatment and procrastination. In most instances the patient should be ready for operation within an hour or two after hospital admission. The operation consists in simple removal of the appendix, usually through a muscle-splitting right lower quadrant incision (McBurney) and closure without drains.

The principles of treatment of appendicitis with localized peritonitis or with diffuse peritonitis are the same as in other infections

bosis or pneumonia is risky. The need for ruling out these conditions before proceeding with the treatment for appendicitis or any other condition should be obvious.

Acute pancreatitis is infrequently a source of confusion. This condition can be diagnosed, however, if the serum amylase test is obtained immediately. The need for the early recognition of acute pancreatitis lies in the fact that operation is sometimes harmful and may result in death, whereas under conservative treatment the patient's chances for survival are better.

Most problems encountered in the differential diagnosis of acute appendicitis arise from disease processes originating in the alimentary, urinary and female reproductive systems. Less often, disturbances of the vascular, nervous, musculoskeletal or male reproductive systems will cause confusion. Rarely will metabolic disorders, such as diabetes mellitus, Addison's disease and porphyria, mimic acute appendicitis.

The clinical features of perforated peptic ulcer, acute cholecystitis, acute intestinal obstruction, acute regional enteritis and acute sigmoid diverticulitis will be described later (Chapter 21). These conditions must always be considered in the differential diagnosis of appendicitis.

Acute gastroenteritis, often due to indiscretions in the diet, ingestion of food contaminated with pathogenic bacteria or alcoholism, may be confused with acute appendicitis. In this condition nausea, vomiting and often diarrhea tend to be more severe than they are in acute appendicitis. The abdominal pain (intestinal colic) and the systemic reaction are also likely to be more severe. Abdominal tenderness is generalized, and there may be diffuse voluntary muscle guarding. Usually there are no localizing abdominal signs such as those seen in acute appendicitis. One must bear in mind, however, that the signs of appendicitis with peritonitis may sometimes resemble those of severe gastroenteritis.

Diseases of the urinary passages, such as right-sided renal or ureteral colic due to stone, acute pyelitis or pyelonephritis, can simulate acute appendicitis. The location and radiation of the pain arising in the urinary tract is commonly located in the lumbar area, the flank or the iliac fossa and radiates into the external genitalia and the thigh. There may be right lower quadrant tenderness, but muscle rigidity is absent because the disturbance arises in the retroperitoneal area. The urine often contains gross or microscopic blood and the plain film of the abdomen may show a stone. Intravenous pyelography

tenderness in the right lower quadrant. Laboratory studies are generally not helpful, but the stool should be examined for parasitic infestation.

A barium enema of the colon often reveals a partial filling or a nonfilling of the appendix. On fluoroscopy the tenderness may be discretely localized to the appendical area.

Patients who present this picture should be carefully studied to exclude other causes for abdominal pain. When no other cause can be established and the pain interferes with normal activity, the patient may be considered to have chronic or recurrent appendicitis and an appendectomy is in order.

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of the peritoneal cavity. Any site of continuing contamination must be removed or closed if possible, the exudate must be evacuated or drained, the infection must be controlled, the gastrointestinal tract must be kept at rest and prevented from becoming distended, and the patient's general condition must be supported. Some patients who have well-localized appendical abscesses, who clinically appear to be well able to cope with the infection and who, under observation, show signs of progressive improvement are sometimes treated without immediate operation. In these instances, if the abscess resolves, "interval appendectomy" in about six weeks is usually advisable because of the danger of recurrent appendicitis.

Moribund patients seen in the late stages of diffuse peritonitis due to a ruptured appendix require a period of intensive resuscitative treatment, including the administration of fluids, electrolytes, blood and antibacterial drugs and tube decompression, before being subjected to the stress of anesthesia and operation.

Recovery from operation for simple acute appendicitis is generally *rapid and uneventful. The patient is ready to leave the hospital* in three or four days and can return to the surgeon's office for the required wound care. When extra-appendical extension is present, the postoperative care is more complicated. The possibility of paralytic ileus, wound suppurations and intraperitoneal abscesses should be kept in mind. A close watch should be maintained for the development of intraperitoneal abscesses. These appear most commonly in one of three regions: (1) the right iliac fossa, (2) the minor pelvis, pouches or culdesacs; and (3) the subphrenic area on the right. Regarding the latter, someone has suggested an appropriate reminder: "Pus somewhere; pus nowhere; pus under the diaphragm."

Chronic Appendicitis

Pathologically, it is often difficult to demonstrate changes which can account for the ill-defined symptoms often considered to arise from a chronically diseased appendix. The symptoms may include: vague, recurrent midabdominal pain; varying degrees of right lower quadrant pain; tenderness without muscle spasm; occasional nausea with or without vomiting, and often a tendency to constipation. Sometimes the only symptom is a "stitch in the side", at other times it is a dull ache. Often the patient is a child or young person The physical examination may reveal nothing beyond mild to moderate

CHAPTER 16

The Lower Alimentary Canal

THE COLON

THE COLON is that portion of the large intestine which extends from the ileocecal valve to the rectum. It consists of the cecum and the ascending, transverse, descending and sigmoid divisions. The ascending and transverse divisions are continuous at the hepatic flexure, and the transverse and descending divisions at the splenic flexure. The splenic flexure usually lies more cephalad than the hepatic flexure. The posterior surfaces of the cecum, the ascending and descending divisions, are devoid of peritoneum and lie in apposition and are fixed to the retroperitoneal structures. The transverse and sigmoid divisions are each suspended by a mesentery, called the "transverse mesocolon" and the "sigmoid mesocolon," respectively. These divisions lie within the peritoneal cavity. The greater omentum is suspended from the transverse colon.

The external surface of the colon has several distinguishing features, including three longitudinal smooth-muscle bands (taenia coli), pouchlike protrusions between the longitudinal bands (haustra) and numerous vascular fat tabs (appendices epiploicae).

The cecum is a large, thin-walled distensible pouch, located in the right lower quadrant; it communicates with the ileum through a bivalved aperture located on its medial wall, the ileocecal valve. The vermiform appendix enters the cecal tip (caput coli).

The ascending and transverse divisions are of smaller diameter than the cecum; and, in fact, the large bowel becomes progressively

REGIONAL ILEITIS (*see under* OTHER DISEASES, p. 392)

APPENDICITIS

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The ascending and *transverse divisions are of smaller diameter* than the cecum; and, in fact, the large bowel becomes progressively

narrower until it reaches the rectum. In other respects the divisions are structurally similar.

From many viewpoints the large bowel may be regarded as a dual organ, the right colon and the left colon, joined in the middle of the transverse segment. Some of the differences between the right

TABLE 17.—SOME DIFFERENCES BETWEEN THE RIGHT AND LEFT SIDES OF THE COLON

	RIGHT COLON	LEFT COLON
Division	Cecum, ascending colon and proximal transverse colon	Middle and distal transverse colon, descending colon, sigmoid and rectum
Embryologic derivation	Midgut	Hindgut
Blood supply	Superior mesenteric artery	Inferior mesenteric and hemorrhoidal arteries
Function	Absorption of water and salts	Storage and excretion
Fecal content	Liquid or semiliquid, heavily contaminated	Semiliquid or formed stool
Typical lesion	Bulky, ulcerating carcinoma, which is not usually obstructive	Annular "napkin ring" lesion which encircles and obstructs the bowel
Clinical syndrome	1. Dyspepsia 2. Anemia and weakness 3. Mass in right lower quadrant 4. Obstructive symptoms (uncommon)	1. Change in bowel habit 2. Partial intermittent or partial and progressive large-bowel obstruction 3. Blood in the stool, occult or gross
Diagnostic considerations	1. History and physical examination 2. Blood studies 3. Stool examination 4. Barium enema	1. History and physical examination 2. Rectal examination, proctoscopy and sigmoidoscopy 3. Biopsy 4. Stool examination 5. Barium enema
Treatment	1. Bowel preparation 2. Resection of right colon with ileocolostomy 3. In acute obstruction, decompression by colostomy or cecostomy, subsequent resection	1. Bowel preparation 2. Resection with end-to-end anastomosis 3. In rectum, abdominoperineal resection with colostomy 4. In acute obstruction, colostomy decompression; subsequent resection

and left halves of the colon are summarized in Table 17. Of importance to an understanding of this subject is the fact that the right half of the colon is concerned with the absorption of water from the liquid or semiliquid material which enters it. This material is highly infectious, very irritating, and easily diffusible when it escapes from the bowel. Being liquid, it will pass through narrowed areas of bowel where solid material could not pass.

The left side of the colon is concerned primarily with conduction of the partially formed stool into the rectum. Little absorption occurs in the left colon. Many of the bacteria die during transit from the right to the left colon; but the bowel contents are still infectious, although less so than in the right half.

The colon is not essential for life. Removal of the left half produces no serious physiologic alterations. Removal of the right half is sometimes followed by temporary loose stools, owing to loss of the large absorbing surface. Total removal of the colon may cause temporary metabolic imbalances due to loss of fluid and electrolytes from the ileum but is otherwise compatible with good health.

The colon derives its blood, lymphatic and nerve supply from two sources, in keeping with its embryologic origin. The right segment (midgut derivative) receives blood from the superior mesenteric artery; its venous blood drains into the portal system via the superior mesenteric veins; the lymph channels pass to the mesenteric nodes along the superior mesenteric artery branches. The autonomic supply of the right colon includes splanchnic fibers from the lower thoracic and upper lumbar cord (T9 to L1), motor fiber from the vagus, and intrinsic elements. Pain caused by disturbances in the midgut segment including the right colon is usually referred to the midzone of the abdomen.

The left half of the colon (hindgut derivative) receives its arterial supply from branches of the inferior mesenteric artery. Venous blood drains via the inferior mesenteric veins to the portal system, and the lymphatic drainage parallels the arterial supply. The innervation of the left half of the colon includes the lumbar splanchnics from L2 and L3 and the pelvic nerves in the distal part. Pain from the left segment is usually referred to the lower zone of the abdomen.

Diseases of the colon lead to altered bowel function; the appearance of blood, mucus or pus in the stool; abdominal pain, distention; obstruction; and secondary systemic changes, including impaired

nutrition, weight loss, weakness and anemia. The symptoms are due to local irritation, inflammation or ulceration, partial obstruction, blood loss and possibly absorption of "toxic" products of bacterial action, especially in lesions of the right side of the colon. Many diseases of the colon follow a chronic course, but all may exhibit acute episodes.

Cancer of the Colon

According to Rankin and Graham, "not more than one fourth of the lesions encountered in the large bowel are so situated as to require diagnostic measures not available to every physician." Cancer of the colon (and in this discussion, including the rectum) is a common disease. It is more frequent in males than in females, and it appears with increasing frequency after the age of forty. In comparison with many other malignant diseases, cancer of the colon grows slowly and metastasizes late. Clinically, there is often a long silent interval; then apparently minor disturbances, commonly ascribed by the patient to "piles," "colitis" or "intestinal flu," appear as the tumor progresses. Despite the long silent interval, the prognosis for cure in cancer of the colon is more favorable than for most cancers of the alimentary tract.

Most of the failures in the treatment are due to delay all along the line: the patient fails to seek medical attention when he should; the physician fails to recognize the significance of the patient's complaints or to examine the patient. Even after the diagnosis is made, the patient may fail to secure treatment. After such delay, many patients have become incurable.

Malignant tumors of the colon are predominantly adenocarcinomas. They are found in all segments of the large bowel, but fully 75 per cent arise in the sigmoid and rectum. Cancers of the left side of the colon (Fig. 73, *B*) are generally annular, stenosing and obstructing (napkin ring) and ulcerate to some degree. Cancers of the right side of the colon (Fig. 73, *A*) tend to be more bulky, to grow into the lumen and to ulcerate extensively (cauliflower). They do not significantly constrict the bowel or impede the flow of the liquid bowel content.

All cancers of the colon spread by invasion and metastasis. When the peritoneal coat becomes infiltrated, extracolonic extension of the tumor has probably occurred. Dissemination to the mesenteric lymph

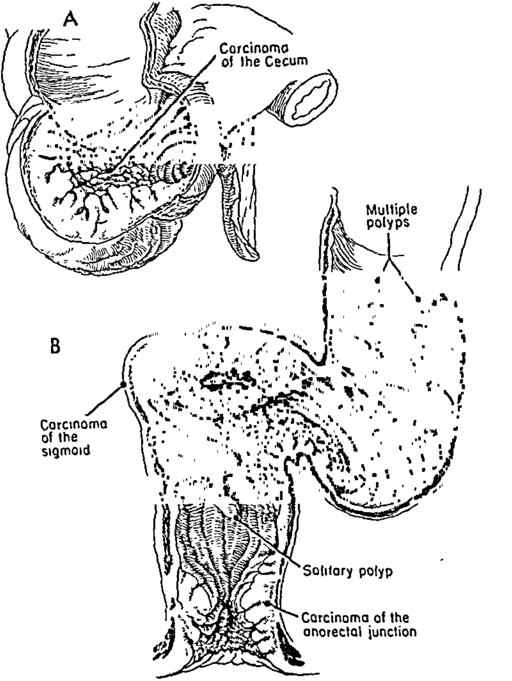

FIG. [illegible]
A, cecal c[illegible]
Obstructi[illegible]
obstruction. Note the annular, constricting character of these tumors. Adenomatous polyps are also present in the rectum and sigmoid.

nodes and to the abdominal nodes occurs with the progression of the disease Blood vascular spread may occur either early or late through the portal system to the liver.

In common with other hollow organs, the large bowel can physiologically compensate and maintain function in the face of a slowly developing obstruction. The smooth-muscle cells become hypertrophied in response to increased work, and normal transit of the fecal stream is thereby maintained for an indefinite time. When the obstruction eventually becomes almost complete, the bowel wall dilates and decompensates. This mechanism tends to delay the onset of obstructive symptoms and favors growth and dissemination of the cancer.

Clinical Picture.—The symptoms of carcinoma of the right and of the left colon are as follows:

Carcinoma of the Right Colon.—Rankin classifies the symptoms from carcinoma in the right colon under three major groups: (1) "dyspepsia," usually diagnosed as chronic appendicitis or chronic cholecystitis, (2) a group which is notable for "toxic absorption" from an extensive ulcerating lesion and (3) a group in which the tumor is accidentally discovered in the right iliac fossa.

The first group of symptoms are indefinite. There may be cramping pain over the lesion and a feeling of fulness after eating, which is associated with a desire to defecate. Diarrhea, blood and mucus in the stools are late signs.

Anemia may be the sole presenting sign. The severity of the anemia may be in direct relation to the size of the lesion, and it is sometimes present before the carcinoma has ulcerated. Early ulceration is the rule because the malignancy is bathed by an irritating and infectious fluid.

Carcinoma of the Left Colon.—The indefinite nature of the early symptoms should be emphasized. Blood in the stool is always significant. The first evidence of obstruction is usually a definite, but ever so slight, change in bowel habit which occurs intermittently and is followed by periods of apparent normalcy. There may be alternating constipation and diarrhea or progressive constipation and discomfort and tenderness in the left lower quadrant.

Acute distention of the cecum is responsible for what may be a misleading localization of abdominal pain to the right lower quadrant. An obstruction in the left colon and a competent one-way ileocecal sphincter convert the large bowel into a "closed loop"

which is unable to decompress itself. This is the mechanism which leads to rupture of the cecum in neglected cases of large-bowel obstruction.

DIAGNOSTIC CONSIDERATIONS.—In order to make the diagnosis of large-bowel malignancy, it is necessary to think of this malignancy. A presumptive diagnosis of carcinoma of the colon is warranted in

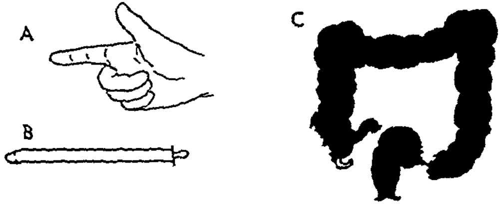

FIG. 74.—Simple diagnostic measures used in detecting cancer of the rectum and colon. A, digital palpation. B, sigmoidoscopic examination. C, x-ray, barium enema.

the following instances, and a careful investigation of the large bowel in search for a carcinoma is indicated in each case:

1. If there is a persistent change in bowel habit.
2. If there is blood in the stool or bleeding from the rectum.
3. If there are persistent vague complaints, referable to the abdomen.
4. If there is obstruction of the large intestine.
5. If there is a hypochromic anemia of obscure origin.

The two major errors on the patient's part are in ascribing his bleeding to "piles" and placing his trust in cathartics if he is "constipated" or "irregular." The great error of the physician is the sin of omission and oversight. The doctor, too, may be satisfied with the explanation that bleeding from the rectum is due to hemorrhoids, and the importance of indefinite symptoms may be overlooked by both the unwary or the negligent doctor and the unknowing patient.

It would be inexcusable to omit examination if the patient had any of the above complaints. The investigation should include digital palpation, sigmoidoscopy and x-ray of the colon (Fig. 74).

FIG. 75.—Barium enema for carcinoma of the sigmoid colon. Note the irregular, partially obstructing filling defect which lies near the rectosigmoid junction. The transverse colon is seen in the upper portion of the photograph, and the ascending colon and cecum are to the left. No barium has passed into the terminal ileum, which suggests that the ileocecal valve is competent, i.e., normally prevents reflux of material from the cecum into the ileum.

It may be difficult to establish the diagnosis if the lesion is beyond the reach of the sigmoidoscope. Roentgenologic visualization of suspect lesions in the lower sigmoid and rectum is uncertain and unreliable. The barium enema is a valuable diagnostic aid in the study of the remainder of the large bowel (Fig. 75). It should be remembered that repeated examinations may be necessary. It may be impossible to differentiate between carcinoma and diverticulitis on the basis of the x-ray evidence. The double-contrast method of colonic examination (air-contrast enema) may be especially useful in the

Fig. 76.—A portion of the colon and rectum excised from a patient with multiple carcinomas and diffuse familial polyposis. Note the cauliflower-like masses in the middle and lower part of the photograph and the variation in the size of the adenomatous polyps, all of which are potentially malignant.

diagnosis of diffuse familial polyposis (Fig. 76), a notorious malignant precursor.

A few precautionary measures which should precede x-ray examination of the colon are important: (1) It is necessary to cleanse the large bowel carefully in order to get satisfactory roentgenograms. (2) The patient should be sigmoidoscoped before barium is introduced into the colon by rectum. (3) The suspicion of obstruction of the large intestine is a contraindication to the administration of barium by mouth.

TREATMENT.—To cure the patient, it is necessary to remove the tumor and the areas to which it may have spread. The operation is generally an extensive one. Cancers of the right colon usually require excision of a short segment of ileum, the cecum, the ascending colon

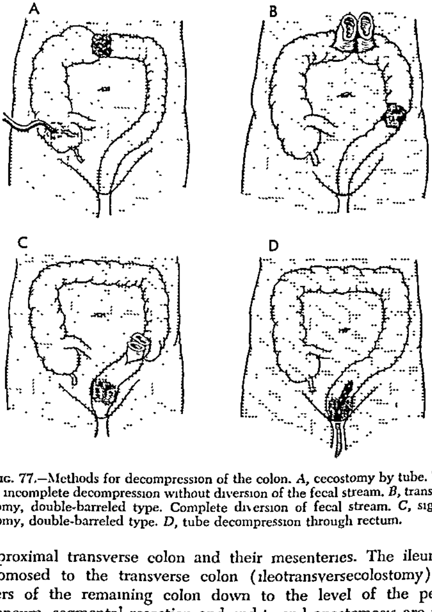

FIG. 77.—Methods for decompression of the colon. *A*, cecostomy by tube. Temporary incomplete decompression without diversion of the fecal stream. *B*, transverse colostomy, double-barreled type. Complete diversion of fecal stream. *C*, sigmoid colostomy, double-barreled type. *D*, tube decompression through rectum.

and proximal transverse colon and their mesenteries. The ileum is anastomosed to the transverse colon (ileotransversecolostomy). In cancers of the remaining colon down to the level of the pelvic peritoneum, segmental resection and end-to-end anastomosis are usually required Cancers at or below the pelvic peritoneum require resection of the sigmoid colon, rectum and anus (combined abdominoperineal resection) with establishment of an abdominal colostomy.

The preoperative care and the postoperative care are extremely important. Special measures are required during the preoperative period to insure a clean and relatively sterile bowel. This is accomplished by the administration of cathartics, a low residue diet and antibacterial agents. The preparation takes approximately five days, Sulfasuxidine,® broad-spectrum drugs or neomycin are commonly used. Enemas are frequently unnecessary.

When there is associated obstruction, the treatment is more complicated. In complete obstruction a preliminary operation becomes necessary for decompression and cleansing of the bowel (Fig. 77). Either a colostomy in the transverse colon or a cecostomy is established. Suction-decompression with a long intestinal tube is generally ineffective in large-bowel obstruction because the one-way action of the ileocecal valve (competent valve) in a majority of individuals prevents passage of gas and fluid from the colon to the small bowel. When the bowel has been cleansed satisfactorily (usually seven to ten days), the second stage, or definitive, operation is undertaken. The colostomy is closed later at a third stage. Most cecostomies close spontaneously.

The management of any patient with a colostomy poses special problems. The patient has no voluntary control of bowel function; and he is plagued by soiling, odors and inopportune movements. These disturbances can be minimized by the use of a fitted colostomy appliance and by establishing a routine type of colostomy care. The essential elements of colostomy care include: a constipating diet, regular colostomy irrigations and drugs, when necessary, during the early period. The most effective agents for the control of loose movements are: paregoric and kaolin, pectin and bismuth mixtures.

A colostomy is often required to relieve an obstruction caused by an incurable cancer. While a colostomy provides temporary palliation, the subsequent course of the unfortunate patients is poor. All other measures available for the relief of pain and disturbed bowel should be judiciously used. Irradiation is not effective.

Chronic Ulcerative Colitis

Ulcerative colitis is a chronic disease, of unknown cause, involving the large intestine and sometimes a part of the terminal small intestine. The clinical course in ulcerative colitis is characterized by recurrences and remissions, leading to progressive destruction and

scarring of the colon, with other complications. The complications are somewhat similar to those of gastric ulcer and include perforation, hemorrhage, obstruction and occasionally cancer. Advanced ulcerative colitis also results in severe nutritional depletion.

The cause of ulcerative colitis is unknown. It has been labeled a "psychosomatic" disease by some. That neuroendocrine mechanisms are concerned in its pathogenesis and/or persistence is probable. Allergy has been frequently incriminated. In some patients, colonic infections appear to play a role. The irritant effect of the digestive secretions on the colonic mucosa may be important when increased levels of lysozyme (mucus-destroying enzyme) appear in the bowel. It is possible that ulcerative colitis, as well as peptic ulcer, does not have a single cause but several causes.

The pathologic changes in the bowel are striking. The disease commonly starts in the rectum or sigmoid and extends to involve the entire large bowel. With repeated inflammation and repair, the bowel wall becomes contracted, thickened and shortened. The haustrations are lost, and externally the bowel often resembles a "lead pipe." The mucous membrane is swollen, red and dotted with innumerable ulcers. The ulcers are usually tiny superficial erosions with necrotic, oozing, granulation-tissue craters. The surrounding mucosa is often hyperplastic, and pseudopolyps may develop. Typically, the entire lining of the involved bowel is friable and bleeds freely when rubbed.

Clinical Picture.—The disease usually appears in early adult life, often in the twenties or thirties. There is sudden onset of diarrhea, with frequent loose or watery stools containing blood, mucus, pus and sometimes fragments of tissue The stools may number from three to thirty daily; and some patients actually spend most of their time on the toilet. With the diarrhea, abdominal cramps, tenesmus (frequent urge to defecate), weakness, weight loss, debility and invalidism may appear.

The acute symptoms frequently abate under treatment but tend to recur periodically, and in severe forms of the disease each attack becomes more severe than the previous one. The changes within the bowel become fixed or progressive and often lead to serious local complications. During this time the patient's general condition may deteriorate in spite of all attempts to control the disease.

In advanced ulcerative colitis there are anorectal, large-bowel and systemic complications. The anorectal complications are in large

measure due to diarrhea and irritations. They include: prolapsed and thrombosed hemorrhoids, cryptitis, fissure, fistula and anal stenosis. The serious large-bowel complications include: partial obstruction, massive hemorrhage, perforation and malignant degeneration. The systemic disturbances include: malnutrition, anemia, hepatic cirrhosis, rheumatoid arthritis, infectious gangrene of the skin (pyoderma gangraenosum) and psychoses.

The diagnosis of chronic ulcerative colitis is suggested by the patient's history. On sigmoidoscopic examination the mucous membrane is found to be diffusely ulcerated and bleeding. Smears and cultures of the exudate usually fail to reveal specific pathogens such as those which cause bacillary or amebic dysentery. Barium studies of the colon reveal a narrowed, shortened, smooth tube. The haustral markings are absent. If biopsy is essential, the specimen must be taken with great care because the diseased bowel is easily perforated.

TREATMENT.—Treatment of uncomplicated ulcerative colitis includes dietary measures, together with chemotherapeutic, anticholinergic and adrenal steroid preparations.

Surgical treatment is usually reserved for intractable forms and complications. At the State University of Iowa hospital, about 50 per cent of the patients ultimately require some form of operative treatment. The known natural history of ulcerative colitis indicates that there is a high incidence of late complications. The implication of these observations is apparent: consideration must be given to operative treatment before the late stages of the disease develop.

The operative procedures applied are directed toward diversion of the fecal stream and removal of the diseased bowel. Diversion may be accomplished by ileostomy or occasionally by colostomy; removal of the diseased bowel may be accomplished by resection of the entire colon or, in segmental disease, resection of a portion of colon. Surgical treatment is usually carried out in several stages, but in favorable cases a single-stage operation may be indicated.

The preoperative and postoperative care of the patient is all-important. Operation is generally preceded by a period of intensive preparation, including the repair of nutritional and metabolic deficits, and cleansing of the bowel. Postoperatively, ileostomy care is a special problem. A glue-on plastic or rubber pouch is placed over the stoma immediately after the operation to collect the irritating ileal discharge. The appliance is removed and reapplied at frequent intervals, usually every two or three days.

Ileostomy alone is an inadequate long-term treatment for ulcerative colitis. Frequently there is a clinical remission, but the process remains active in the bowel and the risk of cancer is real. In most instances, therefore, when surgical treatment becomes necessary, plans must be made for the initial or subsequent removal of the colon.

Total removal of the colon with ileostomy is a formidable operation (or series of operations), but the final outcome justifies the treatment. Usually, there is a rapid gain in weight and strength, a return to useful activity and freedom from pain and intractable diarrhea. An ileostomy existence is not ideal, but often there is no alternative.

Diverticulitis of the Colon

Diverticula are true or false, depending on whether all or only part of the layers of the organ of origin are found in the sac. In the colon, false diverticula are common, especially in the sigmoid portion; and they appear with increasing frequency after the age of forty. In the absence of inflammation or other complications, diverticula (or diverticulosis) are generally asymptomatic.

Diverticulosis of the colon is due to herniation of the mucous membrane through defects in the muscular coat. The defects appear to be the apertures through which blood vessels penetrate the muscle layer, and they are usually located at the margins of the taenia coli. The diverticular sac consists of mucosa, a variable amount of fibrous tissue and peritoneum. The tiny neck of the diverticulum predisposes the sac to stasis of its contents and to inflammation or diverticulitis.

The pathogenesis of diverticulosis of the colon is not entirely clear; but it appears to be related to spasm, constipation and other causes of increased colonic tension.

Diverticulitis is produced by mechanical and bacterial irritation. Entrapment of infectious material within a diverticulum results in edema, inflammatory exudate, vascular impairment and necrosis similar in many respects to that encountered in acute appendicitis. This process may lead to perforation, with the formation of a small abscess, or occasionally to localized or diffuse peritonitis. With healing, scarring occurs, the bowel is narrowed and shortened, and often partial obstruction occurs. Ulceration accompanying the inflammatory reaction results in blood in the stool. In most surgical specimens, some of the diverticula will be free of inflammatory changes, others

will be involved in a chronic inflammatory process, and still others will be the site of acute inflammation.

The symptoms of diverticulitis include: lower abdominal pain and tenderness; constipation, diarrhea or alternating constipation and diarrhea; blood, mucus and pus in the stools; distention; flatulence;

FIG. 78.—Diverticulosis with diverticulitis of the sigmoid colon as demonstrated on barium enema study. Note the narrowing, irregularity and saw-toothed appearance of a localized area of the sigmoid colon due to spasm, scarring and inflammation.

chronic indigestion; and not infrequently a tender left lower quadrant mass. In acute perforated diverticulitis the symptoms and signs suggest "left-sided appendicitis." In chronic diverticulitis the clinical picture simulates cancer of the large bowel.

Sigmoidoscopic examination should precede x-ray studies. The diverticular openings are not often seen. The only changes noted may consist of fixation of the wall, blood or pus in the lumen, and in-

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confined to three bandlike areas, as it is in the colon. As indicated above, the rectum has no serosal covering except in the uppermost portion.

The rectum becomes continuous with the anal canal at the *anorectal line* (dentate, pectinate). This zone represents the point of union between the gut and the proctodeum embryologically. The mucous membrane of this region is gathered into longitudinal folds

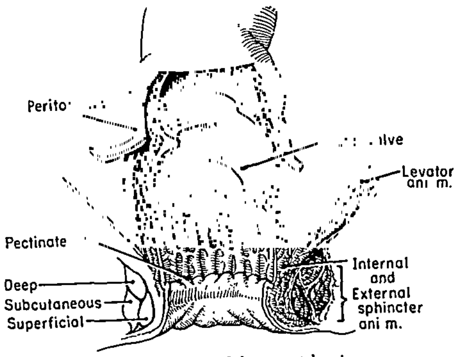

FIG. 79—Anatomy of the anorectal region.

by the action of the sphincter muscles forming the *anal columns.* The terminal bulbous portion of each anal column forms an *anal papilla*. Between each anal column there lies a small pouch, known as the *anal crypt*, below which are located the semilunar *anal valves.* In addition, microscopic-sized *anal glands* or ducts communicate with the anal crypts. The importance of the crypts, glands, valves and papillae of the anal canal lies in the fact that they are concerned in the pathogenesis of most inflammatory conditions which develop in this area (see Fig. 82).

The anorectal line is an important transitional zone. Here the epithelial cells are cuboidal in type, while above, in the rectum, they are columnar, and below, in the anal canal, they are squamous.

ability to introduce the scope to its full length. These findings are not diagnostic.

The diagnosis is usually established by barium x-ray studies of the colon. Characteristically, several diverticula are seen, the bowel is narrowed and has a "saw tooth" appearance, and areas of spasm are observed (Fig. 78). Frequently diverticulitis cannot be distinguished from cancer by means of x-rays.

Treatment of diverticulitis must be individualized. Many patients are managed satisfactorily under a medical regimen. Some will require surgical resection of the diseased bowel. Acute diverticulitis with abscess formation often demands temporary proximal colostomy, sometimes with drainage of an associated abscess. Later the diseased segment can be resected and the colostomy closed.

RECTUM AND ANUS

A large segment of the patients seen by the physician come to him for treatment of anorectal complaints. Often the physician will find that the symptoms are the result of a minor disturbance which is easily treated and cleared up. The net result is many satisfied patients. This is not to imply that serious and complex problems are not encountered, because they are, more often than is commonly realized. Fortunately, most diseases of this area can be diagnosed by simple technics which should be available to every physician. In contradistinction to other portions of the alimentary canal, this is an accessible region.

The rectum extends from the rectosigmoid junction to the anal canal. Except for it proximal end, the rectum lies in the pelvis below the peritoneal reflection. Anteriorly, the rectum is in close relationship to the bladder, prostate, seminal vesicles and urethra in the male, and to the uterus and vagina in the female. Posteriorly, the rectum lies in the sacral hollow. The rectum is fixed, to a variable degree, by its fascial investment, the fascia propria, and the pelvic diaphragm formed by the levator muscles.

In keeping with its storage function the internal diameter of the rectum exceeds that of the proximal large bowel, often measuring 4–5 cm. or more. Its length is variable, but averages about 13 cm.

The inner surface of the midrectum contains three transverse folds, the *valves of Houston*. The longitudinal muscle layer of the rectum spreads out to invest the entire outer wall, instead of being

presence of proximal lymphatic obstruction reversal of flow occurs. Thus, late in cancer of the mid rectum, metastases may be found in the inguinal nodes.

Above the anorectal line the nerve supply consists solely of autonomic fibers, and the rectum is insensitive to usually painful stimuli. The anorectal line and the anal canal, on the other hand, are richly supplied with somatic nerves and are extremely sensitive to painful stimuli.

There is a reciprocal-acting motor innervation to the smooth muscle of the lower bowel. Stimulation of parasympathetic fibers causes increased tone of the bowel and relaxation of the internal sphincter. Stimulation of the sympathetic fibers causes decreased tone of the bowel and contraction of the internal sphincter. The external sphincter is under voluntary control but goes into severe involuntary spasm when irritated.

INVESTIGATION.—The important steps of investigation are:

1. A careful history and complete physical examination
2. Proctologic examination
   - External inspection
   - Digital examination
   - Instrumentation: (*a*) anoscopic, (*b*) sigmoidoscopic
3. Detection of gross or occult blood in the stool
4. Cultures, smears, biopsy
5. Radiographic examination (barium enema, barium-air contrast enema.

HISTORY.—The history should include data regarding the following:

Pain.—Pain is usually the primary complaint. The type of pain, its location, severity and relation to the passage of stools, as well as associated abdominal pain, etc., should be determined.

Blood in the Stool.—The duration, amount and appearance of the blood and its relationship to bowel movements, admixture with stool, etc., should be noted.

Bowel Function.—Changes in the frequency of bowel movements and appearance of the stool should be noted. Remember that medication or changes in diet may mask changes in bowel function. Distention, borborygmus and flatulence may result from local anorectal conditions, colonic disturbances or more distant changes.

Discharge.—Mucoid, purulent or bloody discharge from the anus or perianal area should be noted.

Also important is the point that here the visceral and somatic divisions of the circulation and the nervous system meet. In addition, both voluntary and involuntary muscles which control the outlet of the rectum are located in this area.

The anal sphincter mechanism forms a valve at the anorectal region (Fig. 79). The mechanism consists of two elements: the *internal* and the *external sphincters.* The internal sphincter is a thickened continuation of the circular smooth muscle (involuntary) of the rectum. The external sphincter is composed of striated (voluntary) muscle and contains three divisions, which function as a unit. The divisions from the external surface are: subcutaneous, superficial and deep. The internal sphincter lies above a palpable depression in the anal canal known as the *intersphincteric line,* and the external sphincter lies below. A specialized portion of the levator ani muscle, called the *puborectalis sling,* angulates the anus and rectum anteriorly and contributes to the efficiency of the sphincter mechanism.

The arterial supply of the rectum and anus is derived from the superior, middle and inferior hemorrhoidal arteries. The superior hemorrhoidal artery is the terminal branch of the inferior mesenteric artery. The paired middle hemorrhoidals come off the hypogastric arteries, and the inferior hemorrhoidals come off the internal pudendal branches of the hypogastric.

The veins of the rectum and anus follow a similar pattern. There are: a single superior hemorrhoidal vein; paired middle hemorrhoidal veins, which drain into the portal system; and paired inferior hemorrhoidal veins, which drain to the systemic veins. There are two venous plexuses of the rectum. The submucosal venous plexus, which lies above the anorectal line, is the site of internal hemorrhoids and is drained by the superior and middle hemorrhoidal veins; the subcutaneous plexus, which lies below, is the site of external hemorrhoids and is drained by the inferior hemorrhoidal veins. The plexuses have many intercommunications and, in portal vein obstruction, may serve to shunt blood into the systemic circulation.

The lymph channels and lymph nodes of the rectum parallel the arterial supply. Above the anorectal line the lymph flow is predominantly toward the lymph nodes of the mesentery and pelvic wall, from below the anorectal line to nodes in the inguinal region. It is helpful to conceive of the direction of lymphatic flow (and spread of cancer) from the rectum as being upward, outward and downward, depending upon the level of the rectum involved (Fig. 80). In the

ower bowel and a co-operative patient are essential. Attention to ertain details will aid in securing the patient's co-operation; the ature of the procedure should be explained; and every effort made o avoid pain, discomfort and embarrassment.

The examination should be unhurried, orderly and complete. All endoscopic examinations are potentially dangerous. The sigmoido-

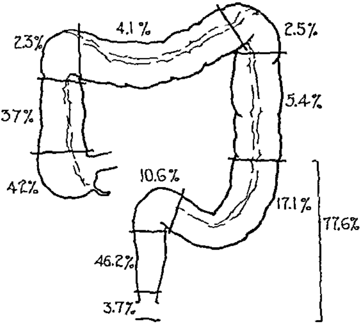

FIG. 81.—Distribution of cancer of the colon, rectum and anus according to segments. Note that about three fourths (77.6 per cent) are in the lower accessible segments. (From Jackman, R. J., *et al.*: Carcinoma of the large intestine; diagnostic errors in relation to location of the lesion, J.A.M.A. 134:1287, 1947.)

scope is always introduced under direct vision after the sphincter area is passed. It may be impossible to introduce the instrument to its full length (25 cm.) because of pain, spasm or fixation of the colon. Under these circumstances the examination should be terminated. Later it may be repeated under more favorable conditions, or the area may be studied by x-ray.

The order of the examination is as follows:

External Inspection.—Look for skin changes, external hemorrhoids, fissure, fistula or other changes.

Digital Palpation.—Note the sphincter tone, tenderness, masses, induration, etc. Feel the prostate in the male, and the posterior aspect of the female pelvic organs. Note the character of the feces,

Protrusion.—Protrusion may suggest hemorrhoids or anorectal prolapse.

Local Irritation and Itching.—Suggests local anorectal or systemic disturbances with associated skin changes.

Previous Anorectal Conditions.—These may be related. Details of

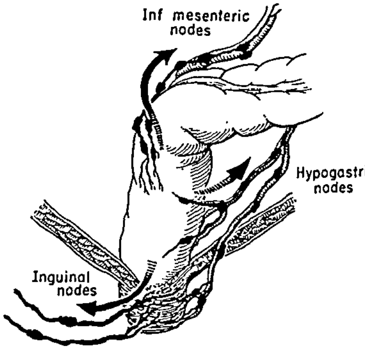

FIG 80.—Lymph drainage of the lower sigmoidal, rectal and anal areas. Note distribution of lymphatics in three zones, upward, outward and downward, respectively. There is, however, considerable overlapping of zones.

any previous treatment, including operation, are helpful in the evaluation.

The history may be of value in suggesting the diagnosis but usually serves only to indicate the need for further investigation.

EXAMINATION.—The position or positions used in examination will depend somewhat on the facilities available and the condition of the patient. A proctoscopic table provides ideal conditions, but a special table is unnecessary for satisfactory examination. The knee-chest position will give good exposure, although it may not be so comfortable or convenient for the patient or the examiner. The lateral (Sims) position may be used when pregnancy, weakness or other complications make the knee-chest position inadvisable.

Adequate lighting, good instruments, proper positioning, a clean

lower bowel and a co-operative patient are essential. Attention to certain details will aid in securing the patient's co-operation; the nature of the procedure should be explained; and every effort made to avoid pain, discomfort and embarrassment.

The examination should be unhurried, orderly and complete. All endoscopic examinations are potentially dangerous. The sigmoido-

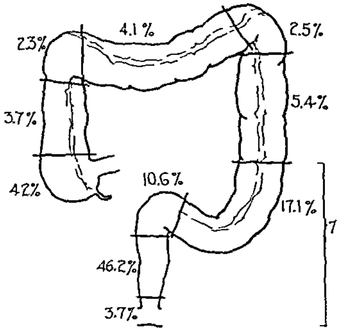

FIG. 81.—Distribution of cancer of the colon, rectum and anus according to segments. Note that about three fourths (77.6 per cent) are in the lower accessible segments (From Jackman, R. J, *et al.*: Carcinoma of the large intestine; diagnostic errors in relation to location of the lesion, J.A.M.A. 134:1287, 1947.)

scope is always introduced under direct vision after the sphincter area is passed. It may be impossible to introduce the instrument to its full length (25 cm.) because of pain, spasm or fixation of the colon. Under these circumstances the examination should be terminated. Later it may be repeated under more favorable conditions, or the area may be studied by x-ray.

The order of the examination is as follows:

External Inspection.—Look for skin changes, external hemorrhoids, fissure, fistula or other changes.

Digital Palpation.—Note the sphincter tone, tenderness, masses, induration, etc. Feel the prostate in the male, and the posterior aspect of the female pelvic organs. Note the character of the feces,

especially with respect to the presence or absence of blood. If grossly negative for blood, test for occult blood.

Anoscopic Inspection.—Look at the anal canal and lower rectum, especially the anorectal line, the crypts, papillae and hemorrhoidal area.

Sigmoidoscopic Inspection.—Pass the instrument through the anal sphincter, remove the obturator and make the examination under direct vision. Air insufflation may be unnecessary. If used, only sufficient air to allow passage of the instrument should be introduced. Suction-aspiration will aid in the removal of fluid and feces for clearer inspection. Biopsy can be performed, cultures taken and polyps destroyed at this time.

Barium Enema.—X-ray studies are necessary for completeness. Barium enema studies, including fluoroscopy and films of the filled and emptied colon, supplemented by air injection (air-contrast) studies may be obtained. Sigmoidoscopy should generally precede barium studies of the colon.

About three out of four cancers of the large bowel are located in the rectum or lower sigmoid (Fig. 81), where they can be felt or seen. The rule that no physical examination is complete without digital palpation of the rectum is based on sound clinical experience. Furthermore, since the rectum and lower sigmoid, because of their size and tortuosity, are relatively blind spots to x-ray examination, direct inspection of this region is most important.

Common Surgical Conditions of the Anorectal Region

An *anal fissure* is an acute or chronic ulceration in the anal canal. It usually results from an injury to the mucous membrane produced by the passage of hard stool over a vulnerable area in the anal ring. The most vulnerable area is located in the posterior commissure, where the sphincter fibers cross and are attached to the anococcygeal ligament. The abrasion results in a vicious cycle of infection, spasm and a painful chronic ulcer. An associated fibrous-tissue response in the anal skin just beyond the fissure produces an external skin-covered mass or "sentinel pile," which protrudes from the anal canal.

The symptoms of anal fissure are due to the ulcer and associated muscle spasm. The pain may be continuous but is more severe with movement of the bowels. Small amounts of blood appear on the stool. There may be a mucopurulent discharge, together with anal itching, constipation, backache and general irritability.

The diagnosis can usually be made on inspection. Pain and muscle spasm make endoscopic examination difficult. A topical anesthetic may be required. After a short period (a few days) of local treatment, a more adequate examination can be performed.

Sitz baths, suppositories, ointments, mineral oil and bulky laxa-

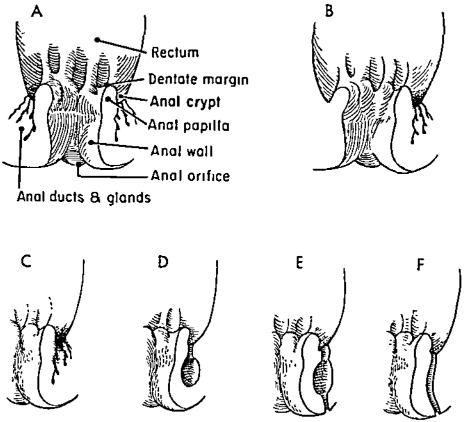

FIG. 82 —Pathogenesis of cryptitis, perianal abscess and anal fistula. *A*, normal relationships *B*, stagnation of feces in an anal crypt. *C*, inflammation of crypt and anal glands. *D*, perianal abscess. *E*, rupture of perianal abscess. *F*, anal fistula. (After Nesselrod.)

tives usually give relief from pain and spasm and lead to partial or complete healing. Operation is required in the chronic stages of anal fissure. The operation consists in excision of the fissure, the crypt and the sentinel pile. Muscle spasm is released by cutting the subcutaneous portion of the external sphincter or by dilating and paralyzing the sphincter (divulsion). The wound in the anal canal is left open and allowed to heal by granulation.

An *anal fistula* is an abnormal communication usually located between an anal crypt and the skin. This common, chronic condition

results, as a rule, from cryptitis. Diseases such as ulcerative colitis, amebic dysentery, lymphopathia venereum, intestinal tuberculosis and radiation proctitis predispose to anal fistulas (Fig. 82). The condition begins with stasis and infection in the anal crypts and/or glands and formation of a perianal abscess. Eventually the abscess ruptures or is incised, and a tract is formed between the anal crypt and the perianal skin. Bacteria, secretions and feces enter the tract and keep it open by constantly "feeding" it. There is also some tendency to epithelial ingrowth at both ends of the tract, which interferes with healing. The patient often gives a history of a perianal

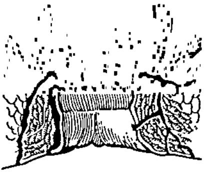

FIG. 83 (*left*).—Anal fistulas. The internal opening of the fistulous tract is usually located in an anal crypt and the external opening in the perianal area. Three types of fistulas are depicted according to the relationship of the tract to the anal sphincter, that is, the tract may pass inside, outside and through the sphincter muscles.

FIG. 84 (*right*).—Internal and external hemorrhoids. Note their relationship with the anorectal line.

"boil" which ruptured, healed and then re-formed. Pain may not be a prominent symptom. Usually the annoying discharge, discomfort and itching bring the patient to the physician.

Anal fistulas are recognized by the presence of an external opening which communicates with an internal opening located in an anal crypt (Fig. 83). A malleable surgical probe can sometimes be passed through the tract, or dilute methylene blue solution can be injected into it. The fistula may be simple and direct, or tortuous, branching and complicated. The tract or tracts can pass inside, through or around the anal sphincter. Occasionally, both the external and internal openings are located within the anal canal or rectum. When fistulas involve the deep portions of the sphincter, special operative procedures are required to preserve anal continence.

Operative treatment is required for the cure of chronic anal

fistulas. Incision, or in some cases excision, is indicated to allow healing by granulation. The wound is not sutured because there is danger of invasive infection; it is left open and allowed to "heal from the bottom." During the postoperative period, frequent inspection is necessary to insure healing without a "bridging-over" of the skin

Hemorrhoids (Fig. 84) are varicosities of the anorectal veins. External hemorrhoids are covered by skin and develop from the external hemorrhoidal plexus; internal hemorrhoids are covered by *mucous membrane* and develop from the internal hemorrhoidal plexus. Combined internal and external hemorrhoids are common. The development of hemorrhoids appears to be related to a local increase in venous pressure from impaired venous flow due to chronic constipation, pregnancy, portal obstruction, pelvic or rectal tumors and other conditions which favor venous stasis. As in the case of varicose veins of the lower extremities, there are probably other predisposing congenital or acquired factors involving the strength of the mesodermal supporting tissues, the venous valves, etc.

Bleeding is the outstanding symptom. Ulceration and thrombosis of hemorrhoids also occurs, resulting in pain, often of severe degree. There may be associated prolapse, discharge and perianal itching.

In mild cases, local measures, including a nonconstipating diet and the avoidance of conditions which cause sudden increases in venous pressure, may be adequate. For bleeding, the injection of small amounts of sclerosing solution into the perihemorrhoidal area (not into the hemorrhoid) has been widely used. Usually such injections give temporary relief. The surgical treatment of choice is excision of the hemorrhoids. The objective of the operative treatment is removal of the hemorrhoids while preserving the normal skin-mucosal relationship and normal sphincter function.

The postoperative care requires attention to wound healing and the prevention of scarring or stenosis of the anal canal. In some cases, digital dilatations are required. Usually, neither restriction of the diet nor prolonged bed rest are necessary.

Cryptitis is an inflammation of the anal crypts and anal glands which often leads to complications. The anatomic features of the anorectal line predispose this area to retention of infectious material and inflammation with abscess formation. The localized process is cryptitis (Fig 82). The complications are perianal, perirectal or ischiorectal, according to the location of the abscess.

The treatment of cryptitis constitutes an important prophylactic measure for the prevention of other anorectal complications. The recognition of cryptitis is not difficult. There is pain and anal muscle spasm. Bleeding is uncommon. The anorectal line is usually tender and indurated. On direct inspection the inflamed crypt can often be demonstrated with a hooked probe. Local measures are used to overcome retention and inflammation. It is sometimes necessary to incise the lower border of the crypt, including the anal valve, thus insuring adequate drainage.

Anorectal abscesses, or perianal abscesses which lie near the anal canal, may develop from infection of the skin glands as well as from infections of the anal crypts. Perirectal abscesses generally originate from infection of the anal crypts and lie along the rectal wall. Ischiorectal abscesses are complications of cryptitis, fissure, fistula or other anorectal infections. The abscess is localized to the area between the anus and the ischial tuberosities. There is severe pain, swelling and tenderness. The area must be drained and allowed to heal by granulation. A chronic fistula may ultimately form.

Anal pruritis, a complaint rather than a specific disease, is caused by a variety of local and systemic disturbances. Often the cause is obscure. In severe forms it is characterized by persistent and intractable itching of the perianal area and frequently the genital area. It occurs more commonly in men than in women and is sometimes associated with an overt emotional disturbance. Attention must always be directed toward a possible emotional problem if no local cause for the symptoms can be established.

The skin changes are slight or marked, depending on the severity, the duration and the effects of scratching and medication. Typically the skin is pink, thickened, moist and excoriated.

Anal pruritis can result from any local condition which produces irritation of the skin, such as poor hygiene, diarrhea, chronic anal discharge or fungus or parasitic infections.

Every effort should be made to locate and eliminate all sources of irritation. Factors relating to the general health of the patient must be investigated. Diabetes must always be considered. Many ointments and lotions are available for local application, most of which give temporary relief. Patients in whom no local lesion can be demonstrated and who do not respond to the usual measures are sometimes subjected to more radical treatment, including: irradiation, alcohol infiltration, undercutting (denervation) with excision of the

perianal skin, and tattooing with mercuric salts, all of which give varying degrees of success.

Rectal prolapse is a condition in which a portion of the bowel wall protrudes through the anal opening. The protrusion may consist of mucous membrane only (incomplete prolapse) or of the entire thickness of the bowel wall (procidentia).

Mucosal prolapse is common in adults and is often associated with internal hemorrhoids. In mild prolapse, conservative treatment, such as measures to soften the stool, prevention of straining and sustained reduction of prolapse, often proves adequate. When hemorrhoids are associated, operative treatment may be required.

Complete prolapse is brought on by straining, constipation or diarrhea. It is common in children; less frequent in the adult. Complete prolapse can be recognized by the concentric arrangement of the folds of mucous membrane and palpable two thicknesses of bowel wall where the mass protrudes through the anal orifice. In incomplete prolapse the mass is smaller and is covered by radiating folds of mucosa (as in the spokes of a wheel).

When a complete prolapse is treated conservatively, the response is usually favorable in the infant or child; but radical surgical measures are necessary for severe degrees of prolapse in the adult.

Adenomatous polyps of the rectum and colon arise from apparently normal mucous membrane. They are composed of glandular elements covering a connective-tissue stroma. Microscopically, the epithelium of polyps shows a wide range of changes, from a normal cellular pattern to areas of premalignant and/or definitely malignant changes (Fig. 76, on p. 371). Polyps range from flat (sessile) to pedunculated tumors. They vary in size from 1 mm. or less to 2 or 3 cm. or more. They may occur as single or multiple lesions. Sometimes they are widely separated; at other times, they are diffusely distributed throughout the large bowel.

Patients with untreated adenomatous polyps of the rectum and colon who live long enough will probably develop carcinoma. This prediction is based on observations that (1) with the passage of time this type of polyp tends to undergo malignant changes and (2) the known high incidence of polyps associated with large-bowel cancer.

Often there are no symptoms, and they are found during a routine examination or an examination for an unrelated condition. The more polyps of the colon are looked for, the more often they are found.

Slight bleeding is the only significant symptom. The predisposition to the recurrence of adenomatous polyps is so great that, once the diagnosis is established, it can generally be predicted that the patient will have more polyps. All patients with demonstrated adenomatous polyps of the rectum and colon require frequent checkups (usually 3 to 6 month intervals). This entails sigmoidoscopic and barium (especially air-contrast) studies of the colon.

Large polyps, both pedunculated and sessile, must be considered malignant until proved otherwise. Biopsy excision or biopsy of several areas is indicated according to the gross appearance of the lesion. A complete destruction or removal of the tumor through the sigmoidoscope or through a transabdominal operation is necessary according to the location, size and biopsy findings. If unequivocal malignant changes are present, more radical treatment is indicated. Tiny polyps are destroyed by electrosurgical methods.

Diffuse generalized polyposis is a familial disease which usually appears before the age of thirty. As a rule, there is progressive involvement of the colon and ultimate neoplastic changes. Symptoms are due to mucosal ulceration and interference with normal bowel function. The diagnosis is often suggested by the history of an unusually high incidence of cancer of the bowel in the patient's forebears and is confirmed on examination. Under these circumstances, the physician must recommend examination of all blood-relatives. In some patients, local removal of the polyps and planned regular follow-up observations is possible. For those patients with established diffuse polyposis, the removal of the entire colon is required.

Pilonidal cysts and sinuses are defects of developmental origin located over the sacrococcygeal area. They consist of epithelial-lined, hair-containing cysts or sinuses. They often become infected, producing acute or chronic abscesses. Trauma may precipitate or aggravate the condition. They are seen chiefly in young adults, both male and female.

The pathogenesis of pilonidal sinus has been variously ascribed to faulty invagination of the ectoderm of the median raphae, to maldevelopment of the terminal spinal canal, and to hairs driven into or buried in the skin of the intergluteal cleft. The existence of a pilonidal cyst is suggested by one or more small openings or dimples located in the intergluteal cleft.

Pilonidal cysts complicated by acute infection require warm packs, drainage and antibiotic therapy. Recurrences are common, and the con-

dition tends to become chronic. Complete excision is usually necessary for cure.

Measures Commonly Used in Anorectal Diseases.—*Hygiene of the Perianal Area.*—Hygiene includes: frequent cleansing and drying; avoidance of irritation; and the use of cotton wipes and witch hazel lotion.

Sitz Baths.—The patient sits in a tub of warm water for fifteen minutes, two or three times daily. The temperature of the water may be increased during the course of the bath but need not be "as hot as the patient can stand."

Warm Compresses.—Frequent applications of saline or mildly medicated solutions to perianal area are advised.

Lotions and Ointments (including fungicides).—These often prove helpful.

Suppositories.—The "-caine" preparations (Nupercaine,® Surfacaine,® butacaine, etc.) often produce sensitivity reactions; they should be used cautiously.

Other Measures—Analgesics, sedatives, antibiotics, change in diet and bowel care (see below) should be considered.

Surgical Measures.—These include: Excision, incision, stretching (divulsion) of the sphincter, or division of the subcutaneous portion of external sphincter.

Injection.—Sclerosing, anesthetic, or tattooing solutions are used advantageously by those trained in this field.

Bowel care.—The bowel care requires consideration of—

Diet (bland, low-residue, nonallergic or general)
Lubricants (hygroscopic [methyl cellulose], petrolatum, agar)
Antibiotics (locally, systemically or combined)
Antispasmodics, antiperistaltics (belladonna, opium, etc.)

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CHAPTER 17

The Biliary Tract

THE BILIARY TRACT is a complex system for the conduction, concentration, pressure regulation and delivery of hepatic secretion to the intestine (Fig. 85). The system consists of the right and left hepatic ducts, the common hepatic duct, the cystic duct and gallbladder, the common bile duct and the sphincter of Oddi. The common hepatic duct lies above the union of the cystic duct with the common duct; the common bile duct lies below this union. The proximal common bile duct lies within the free margin of the lesser omentum; the distal portion passes behind the duodenum, then through the head of the pancreas and enters the posteromedial wall of the second portion of the duodenum obliquely. The common duct is about 8 cm. in length and 8 mm. in diameter, or somewhat smaller than a pencil. The proximal or supraduodenal portion of the common bile duct, the hepatic artery and the portal vein are all located in the right border of the lesser omentum, which forms the anterior boundary of the epiploic foramen (Winslow). The first two structures lie anteriorly, the duct to the right and the artery to the left; and the portal vein lies behind.

The gallbladder is a thin-walled, grayish blue, pear-shaped sac which is attached to the inferior surface of the right lobe of the liver. It is about 10 cm. long and has a capacity of roughly 50 ml. Its bulbous portion is called the "fundus," and its narrowed portion the "ampulla." Frequently, overlying the cystic duct there is a diverticulum-like enlargement of the ampulla, known as "Hartmann's pouch." The ampulla of the gallbladder is continuous with the cystic duct, which enters the common duct.

The cystic duct is about 2.5 cm. in length and 3–4 mm. in diameter. It contains mucosal folds, or the spiral valves of Heister, which are

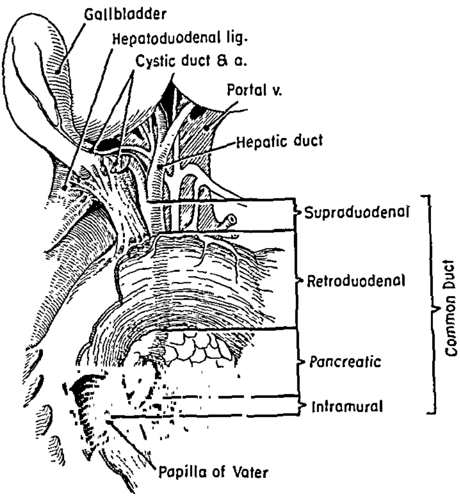

FIG. 85.—Anatomic relationships of gallbladder, bile ducts, duodenum and head of pancreas. The four divisions of the common bile duct and its position with respect to the hepatic artery and portal vein are shown. The most frequent pattern of the duct system and the vascular supply is depicted, but variations from this pattern are the rule rather than the exception.

probably concerned with the regulation of bile flow. The cystic duct, which is often tortuous and has a beaded appearance, passes downward and to the left to join the common duct; and the cystic artery arises from the hepatic artery and passes under the common hepatic duct to the gallbladder. These three structures—common duct, cystic duct and cystic artery—form an anatomic triangle (Calot), the base of which is the cystic artery.

The duodenal papilla (Vater) is a nipple-like projection located within the duodenum about 10 cm. from the pyloric valve, which

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osis); and when complete obstruction to the gallbladder outlet occurs, the bile pigment is absorbed, producing so-called "white bile." Likewise, under conditions of obstruction or inflammation, mucus is secreted in increased amounts. When the mucosa is damaged, the muscularis and serosa are also damaged. Inflammatory cell infiltration, edema, scarring and a variable degree of interference with lymphatic and blood flow develops. Over a period of time the entire wall becomes thickened and contracted and there is loss of normal function.

The function of the gallbladder is integrated with that of the sphincter of Oddi and the duodenum through autonomic and humoral mechanisms. The smooth operation of this system depends on a normal flow of hepatic bile, an open biliary tract, an absorptive gallbladder membrane, a distensible and contractible gallbladder wall, a functioning sphincter of Oddi and a balanced control mechanism. Obviously, there may be many conditions of organic and functional origin which can interfere with the smooth operation of any segment of the system and cause clinical symptoms.

The liver secretes bile continuously, but the output is increased during meals. Before food passes into the duodenum, the sphincter of Oddi is closed, hepatic bile enters the gallbladder and, by a process of absorption of water by the gallbladder, the bile is concentrated up to ten times its original volume. When, after eating, gastric chyme enters the duodenum, a hormone, cholecystokinin, is released into the blood from the duodenal mucosa. This causes the gallbladder to contract and empty its contents into the common duct and duodenum, the sphincter having already opened. Bile (and pancreatic juice) enter the duodenum in spurts as contraction of the gallbladder continues. By the time the stomach has emptied, the gallbladder is also empty. The sphincter now closes, the gallbladder then relaxes and again begins to fill. When the gallbladder has been removed or has been rendered functionless by disease, the mechanism for the concentration and storage of bile is lost and bile flows into the duodenum continuously.

The pathogenesis of biliary tract disease is not completely understood. While there is much evidence to indicate that it results from metabolic disturbances rather than infection, infection often is superimposed, once the disease is initiated. The association of gallbladder disease with aging, pregnancy, obesity and degenerative vascular diseases suggests that disorders of lipid metabolism, especially cholesterol, are important. Gallstones are commonly associated with biliary

represents the end of the common bile duct and the main pancreatic duct. Ordinarily the common bile duct and the main pancreatic duct unite at the ampulla of Vater, a slightly dilated portion of the common duct. The sphincter of Oddi is a ring of smooth muscle fibers which encircles the duct system between the ampulla and the papilla and serves to regulate the flow of bile and pancreatic juice into the duodenum and to prevent regurgitation of fluid from the duodenum into the ducts.

The blood supply of the biliary tract, in common with other foregut derivatives, is derived from the celiac axis. The hepatic artery from the celiac axis gives off several branches to the stomach, duodenum and pancreas, and, via the cystic artery, supplies the gallbladder. It terminates as the right and left hepatic arteries. The hepatic artery is the sole source of arterial blood flow to the liver, and occlusion of this vessel usually leads to liver necrosis and death.

The veins of the biliary system drain to the portal vein. The lymphatics accompany the arteries and drain to nodes around the common bile duct, the celiac axis and the liver hilum, as well as directly to the liver.

The gallbladder and bile ducts receive their nerve supply from the autonomic nervous system through splanchnic and vagal fibers Visceral pain from this area is referred to the upper abdominal zone, and often to the back, the right scapula, the right shoulder and interscapular areas through communications in the cord (T5 to T9).

Variations in the anatomic pattern of the biliary tract are exceedingly common—they may be said to be the rule rather than the exception. Both ducts and blood vessels are subject to anomalies of structure and position. The importance of these variations lies in the fact that too often they are not recognized at operation, and accidental injury to the common duct or to the hepatic blood supply occurs. The danger of injury is minimized if the structures are dissected out and identified before they are ligated or divided. When the common duct is injured, scarring with stricture formation follows and the patient may die or suffer permanent invalidism.

The gallbladder wall is composed of three layers—the mucosa, the muscularis and the serosa. The mucosa is a soft, folded, velvety epithelial membrane which absorbs water, electrolytes, probably some bile salts and possibly cholesterol. Here, too, mucus is secreted into the bile. Disturbances in gallbladder absorption or secretion often lead to precipitation of cholesterol crystals in the mucosa (cholester-

ing response. Special intravenous media (Cholografin®) which afford almost immediate visualization of the gallbladder and duct structures are also available.

When there is disease of the biliary tract, the cholecystogram may show that the gallbladder fills poorly or not at all, that it contains radiopaque or radiolucent stones or that it empties poorly or not at all. Whatever the radiographic findings may be, they must be correlated with the clinical findings before evaluation of the patient's condition can be said to be complete. When the x-ray findings are equivocal, the test should be repeated.

Disturbances of the pressure-regulating function of the biliary tract are common. They may have their origin in the gallbladder, the common duct, the sphincter of Oddi or the regional structures (stomach, duodenum, pancreas, etc.) and may be caused by inflammation, infection, stones, stenosis, tumors or neurogenic (autonomic) influences. Sometimes after cholecystectomy the patient may have persistent complaints or new symptoms which are due to loss of the pressure-regulating function of the biliary tract, or "biliary dyskinesia." In most cases, and provided that there is no organic disease, the common duct gradually dilates and the symptoms disappear.

A number of smooth-muscle relaxing drugs are effective in the control of the symptoms of biliary dyskinesia as well as those of biliary colic due to stone. The spasmolytic drugs include: nitroglycerin, amyl nitrite, papaverine, atropine and, in severe colic, Demerol.®

Biliary tract disease starts in the gallbladder, where it may remain localized; or it may extend to the duct structures, then the liver and sometimes the pancreas. In the gallbladder it leads to pathologic and physiologic alterations of the gallbladder wall and stone formation (chronic cholecystitis with cholelithiasis). If the stones cause complete obstruction of the gallbladder outlet, acute cholecystitis, empyema of the gallbladder or hydrops of the gallbladder results according to the duration of the obstruction and inflammation. Acute obstructive cholecystitis may lead to ulceration, gangrene or rupture of the gallbladder and to formation of a pericholecystic abscess, bile peritonitis or cholecystoenteric fistula.

When the disease process extends to the common duct, stones may be formed within the duct or may pass into the duct from the gallbladder. The duct becomes dilated as a result of partial obstruction and inflammation. Infection is then frequently superimposed.

tract disease and may develop suddenly over a period of months or intermittently in "showers." When precipitation once occurs in the gallbladder, the stones may grow by accretion. It is not difficult to understand the pathogenesis of pigment stones which are often encountered in patients with congenital hemolytic anemia. In this condition, large amounts of bilirubin are excreted in the bile; and with concentration of this substance in the gallbladder, precipitation occurs.

Whatever the underlying causes of gallbladder disease may be, the pathologic changes which follow include: scarring of the gallbladder wall, deposition of cholesterol crystals in the mucosa, stone formation and altered absorption. Stasis and excessive concentration of the bile follows. As the concentration of bile salts is increased, cholesterol goes out of solution and crystallization occurs. A nidus for stone formation thus appears, and soon more cholesterol, bilirubin, calcium or other substances are added.

Gallstones are single or multiple, small or large, smooth or irregular, round or faceted, soft or hard objects. They are brown, black or grayish in color and, when cut and polished, may resemble a precious gem. Some gallstones are composed of many concentric rings, which are, to some degree, an indication of the age and life history of the stones. Usually the stones in any one patient are similar in size, shape and composition.

Gallstones may lie free within the gallbladder lumen or may completely fill it. If they suddenly become impacted in the ampulla or cystic duct, they cause obstruction and precipitate acute cholecystitis. Small stones often pass through the cystic duct into the common bile duct; large stones sometimes erode through the gallbladder and neighboring duodenum or colon, forming a cholecystoduodenal or cholecystocolic fistula. If the stone is unable to pass through the narrowed portions of the bowel (duodenojejunal angle, ileocecal valve, sigmoid colon), intestinal obstruction due to obturation occurs.

X-ray examination of the gallbladder (cholecystography) constitutes the single most important method for the evaluation of gallbladder function and the demonstration of stones. This test depends on the ability of the gallbladder to concentrate and evacuate bile that contains a radiopaque organic iodine compound. The "dye" is usually given orally the night before the examination; and the next morning, films of the gallbladder are made. Following this, the patient is given a fat meal and the films are repeated to observe the empty-

cholecystitis with stones is rapid and the follow-up results are good. The follow-up results are generally poor when little evidence of gallbladder disease and no stones are found at operation. For this reason, cholecystectomy should not be undertaken unless the clinical and x-ray evidence clearly implicate the gallbladder as the source of trouble. As is true in many surgical conditions, the follow-up results are best when the operative and pathologic findings can be definitely correlated with the clinical picture.

Acute Cholecystitis

When the outlet of the gallbladder becomes suddenly and completely obstructed, acute cholecystitis follows. The usual cause of the obstruction is a gallstone impacted in the gallbladder ampulla or the cystic duct. On rare occasions, an inflammatory edema due to infection or irritation of the mucosa by concentrated bile or reflux of pancreatic juice may close the gallbladder outlet even in the absence of stones. When obstruction occurs, there is stasis of bile, mucosal irritation, increased mucus secretion and production of an inflammatory exudate. Infection may then be superimposed. The gallbladder becomes distended, and soon interference with the blood, lymphatic and tissue fluid interchange in its wall occurs. Ischemia, ulceration, necrosis (Fig. 86) and perforation of the gallbladder wall may then develop unless the obstruction is relieved. The obstruction can be relieved spontaneously by displacement of the stone (into the gallbladder fundus or through the cystic duct), by rupture of the gallbladder or surgically by removal of the stone. Frequently, the obstruction persists; and after the initial acute inflammatory reaction (empyema of the gallbladder) has subsided, a chronic condition known as hydrops of the gallbladder appears. In hydrops the gallbladder is often greatly distended, thin walled and filled with water-clear mucus.

Acute cholecystitis is generally a complication of chronic cholecystitis. There is likely to be a history of previous indigestion, upper abdominal distress, qualitative food intolerance and biliary colic. The acute attack appears suddenly, with severe epigastric and right upper quadrant pain and tenderness, often with referred pain to the back and right shoulder, nausea and vomiting, fever and sometimes chills.

There is striking upper abdominal tenderness and muscle guarding, most pronounced over the gallbladder area. The gallbladder is

Soon the liver is involved as a result of cholangitis and back pressure, and a varying degree of biliary cirrhosis develops. Similarly, the process may extend to the pancreas, with the production of chronic or recurrent acute pancreatitis.

Chronic Cholecystitis

Gallbladder disease occurs in both men and women predominantly after the age of forty. It is rare in childhood. Often the symptoms first appear during the period of active sex life of the individual, but serious disturbances do not appear until later in life. Although common in "fertile females, who are fair, fat and forty," the disease is by no means limited to this category. It is usually associated with gallstones, and symptoms result from impaired gallbladder function, bile stasis and disturbed digestion.

The patient complains of upper abdominal distress or pain, together with fulness and belching after meals, which is aggravated by the ingestion of fatty, fried or bulky foods. There is heartburn, nausea and sometimes vomiting of bile. Intermittent attacks of colic, often radiating into the back, shoulder or scapular area may be prominent. Constipation, flatulence and bloating are common.

There are few significant physical findings in uncomplicated cases. Tenderness over the gallbladder region may be all that is found. Generally the gallbladder is not palpable and the liver is not enlarged.

Likewise, there are no noteworthy changes in the blood, urine or stool. Plain x-rays of the gallbladder area may show calcium-containing radiopaque stones, or a cholecystogram may reveal radiolucent stones with absent or poor gallbladder filling.

Surgical treatment is recommended for established chronic cholecystitis with stones. The operation consists of removal of the gallbladder, careful examination of the common bile duct and exploration of the duct, if indicated. The indications for common duct exploration include: (1) existing jaundice or a history of jaundice, (2) palpable stones within the duct, (3) enlargement or thickening of the duct, (4) many small stones in the gallbladder or a large cystic duct which would allow passage of stones and (5) stones or other abnormalities noted in x-ray studies made during the operation (operative cholangiography).

Usually, recovery from cholecystectomy for uncomplicated chronic

In the differential diagnosis of acute cholecystitis, the following diseases should be considered: perforated peptic ulcer, acute intestinal obstruction, acute appendicitis, acute pancreatitis, renal colic and acute coronary thrombosis. If the diagnosis is obscure, special studies may be required and they should be made promptly. The special studies include: plain films of the abdomen and chest, intravenous pyelography, a serum amylase test and an electrocardiogram.

The treatment of acute cholecystitis depends on the general condition of the patient, the local condition as indicated by the clinical findings, and the duration of acute disease. Immediate operation is necessary in some patients; and delayed operation (after the acute process has subsided), in others. In certain, seriously ill patients the operative treatment consists of simple drainage of the gallbladder (cholecystostomy) and removal of gallstones. In general, however, cholecystectomy is the preferred operation and may be performed either shortly after the onset of acute cholecystitis (within forty-eight hours) or, electively, after the acute process has subsided (usually after about six weeks). During all phases of the illness, there is need for the intelligent use of all the important nonoperative measures, including gastrointestinal decompression, parenteral fluids, antibiotic drugs, etc.

Obviously, treatment of acute cholecystitis must be individualized according to the circumstances. However, certain generalizations are possible. Good-risk patients with established acute cholecystitis of relatively short duration (less than forty-eight hours) can be subjected to cholecystectomy. Most patients with established cholecystitis of greater duration should be treated conservatively and subjected to elective cholecystectomy under more optimal conditions later. When, however, while the patient is under observation, the signs and symptoms indicate progression of the disease and that rupture of the gallbladder is impending, or when the diagnosis is in doubt, early operation is indicated.

Common Duct Stones

The possibility of stones within the common bile duct should be considered in all patients who present evidence of biliary tract disease. When present, common duct stones are practically always associated with gallbladder stones; but after removal of the gallbladder, they may be retained within the common duct or may be

often palpable as a globular mass at the right costal margin, with the mass moving on respiration. Murphy's sign of acute gallbladder inflammation is positive. The sign consists of a sudden arrest of inspiration because of pain when the hand is held over the gallbladder area and the patient is asked to take a deep breath.

The urine is normal, except in acute cholecystitis complicated

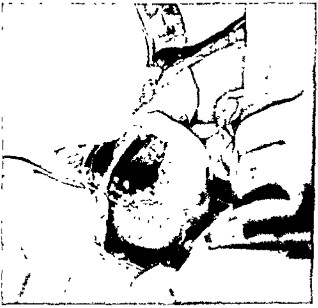

FIG. 86 —Acute obstructive cholecystitis with tension necrosis of the gallbladder fundus. Rupture of the gallbladder has not yet occurred An impacted stone was removed from the neck of the gallbladder, and drainage was established by cholecystotomy The patient made an uneventful recovery.

by dehydration, alkalosis, ketosis or diabetes. It should be recalled that cholecystitis and diabetes mellitus are often coexistent.

The blood findings are those of acute inflammation and are therefore nonspecific. Leukocytes in excess of 15,000/cu. mm with a predominance of polymorphonuclear elements is a common finding. The red cell values are unchanged except as they are influenced by dehydration

Plain x-ray films of the abdomen often reveal radiopaque stones, since about 20 per cent of the patients with gallstones have calcium-containing calculi Plain films will also be helpful with respect to differential diagnosis, to the extent that they often serve to rule out other common causes of acute abdominal pain. Cholecystography is of no value, because the obstructed gallbladder does not fill with dye.

bilirubin and serum alkaline phosphatase, and practically normal hepatic function tests. When the obstruction is persistent, hepatic function becomes more and more impaired. The liver profile ultimately comes to resemble that of diffuse parenchymal disease. The prothrombin level falls, the flocculation tests become positive and the serum proteins, particularly the albumin fraction, are decreased. (See Chapter 18, on the Liver.)

All patients with impaired hepatic function with or without obstructive jaundice should be placed on a short, but intensive, regimen to improve liver function before operation. This should include correction of fluid and electrolyte imbalance, restoration of normal blood values, correction of the prothrombin deficiency, a high intake of carbohydrate, protein and vitamins and the administration of broad-spectrum antibiotics, if indicated.

The surgical treatment consists of removal of stones from the common bile duct and the re-establishment of its patency, together with removal of the diseased gallbladder. The duct is closed around a T-tube drain, the short arm of which is placed inside the duct. X-rays of the duct after the injection of contrast medium are often taken before closure of the abdomen, to demonstrate that the duct has been completely cleared and is open. The T-tube is usually removed in about ten days, and the opening in the duct closes spontaneously.

Cancer of the Gallbladder and Bile Ducts

This is an infrequent type of cancer which is nearly always encountered in patients who have pre-existing biliary tract disease and gallstones. The association suggests that chronic irritation from stones plays a role in the pathogenesis of cancer, but the point remains unproved.

The tumor can arise in any portion of the mucosa of the gallbladder, grow into the wall and lumen and eventually occlude the gallbladder outlet. The tumor may spread to the liver and the common duct directly, as well as to the regional nodes through lymphatics. Usually by the time operation is undertaken, the condition has already become incurable.

The signs and symptoms of carcinoma of the biliary tract are similar to those of benign disease except that in this condition the course is one of progressive deterioration. With obstruction, the gallbladder becomes distended with white bile and tumor. Involvement

formed within it. Thus, patients who complain of persistence or recurrence of biliary tract symptoms months, or years, after cholecystectomy may harbor common duct stones.

Common duct stones are a frequent cause of obstructive jaundice. The stones (or stone) usually pass into the terminal portion of the duct and by means of a "ball valve" mechanism produce intermittent jaundice, with colicky pain, chills and fever, pruritus, light stools and dark urine. If the stone becomes impacted within the duct, the jaundice is likely to be persistent. In some instances the stone is "silent" and painless. Jaundice which results may closely simulate the clinical picture of obstructive jaundice due to cancer of the head of the pancreas. In still other cases, jaundice may not appear because the degree of obstruction is insufficient to cause bile retention. The presence of common duct stones should be suspected from a history of recurrent attacks of pain, fever, chills, and changes in the color of the stool and urine, even in the absence of a history of jaundice.

The findings on physical examination are not remarkable except when there is overt jaundice and hepatic insufficiency. The gallbladder is generally not palpable, because long-standing inflammation and thickening have caused it to become contracted and nondistensible. This is an important point in the differentiation of benign from malignant types of obstructive jaundice (Courvoisier's rule). In malignant obstruction of the common bile duct due to cancer of the head of the pancreas, the gallbladder is thin-walled, distensible and often palpable.

With common duct stones, there is soreness and tenderness in the right upper quadrant. The liver border is firm and extends below the costal margin. Often the sclerae are discolored or "muddy" even in the absence of jaundice. There may be excoriations of pruritus, infrequently vascular "spiders" and occasionally subcutaneous hemorrhages due to the bleeding tendency that accompanies prothrombin deficiency. Weight loss and weakness are usual, but generally not so advanced as when due to malignant disease of the pancreas or bile ducts.

The laboratory studies are important when there is evidence of duct obstruction and impaired liver function. In these conditions the alterations in the urine, stool, blood and liver tests (liver profile) will depend on the degree and duration of duct obstruction and liver damage. Patients with complete common duct obstruction of short duration (weeks) generally have bilirubin but no urobilinogen in the urine, no bile pigment in the stool, moderate elevation of the serum

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of the common duct results in obstructive jaundice. Soon the liver becomes enlarged and nodular. Fever, chills, vomiting and rapid loss of weight and strength follow.

Curative surgical treatment is possible only when the disease is localized to the gallbladder. Not infrequently, conditions are favorable for cure when early cancer of the gallbladder is found in patients operated on for benign disease. Palliative surgical treatment for advanced conditions is unrewarding, and radiation therapy is rarely helpful.

Postcholecystectomy Syndrome

The term "postcholecystectomy syndrome" has been applied to an ill-defined group of symptoms which sometimes appear after cholecystectomy. The symptoms are similar in many respects to those of primary disease of the biliary tract and consist of epigastric distress, especially after eating, fulness, belching and sometimes regurgitation, intolerance to fatty foods, etc. They may be the result of residual disease of the bile ducts (e.g., common duct stone) or dysfunction of the sphincter mechanism (dyskinesia), or due to disturbances beyond the bile ducts (chronic pancreatitis, hiatal hernia of the stomach, peptic ulcer, duodenal diverticuli). In the latter group, removal of the gallbladder could not be expected to relieve all symptoms.

In order that the patient be given the best chances for relief of the distress for which he has consulted the surgeon and submitted to operation, it is necessary that the diagnosis of gallbladder disease be correct, that those diseases which simulate gallbladder disease be excluded, that the diseased gallbladder be removed, that the duct structures be cleared if necessary and that the postoperative care be good. If these conditions are fullfilled, postcholecystectomy symptoms will be few and usually transient.

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CHAPTER 18

The Liver

THE LIVER is the largest organ in the body. It weighs about 1,500 Gm. in the normal adult. Aside from traumatic injuries, abscesses and occasional neoplasms (primary or localized metastatic tumors), most conditions of the liver are not amenable to direct surgical measures. More often the liver is the site of physiologic or biochemical alterations which arise from metabolic, nutritional or infectious causes, bile duct or vascular obstructions and congenital or neoplastic diseases. These states lead to localized or diffuse changes in hepatic structure and function. Thus the liver may be affected by a wide variety of acute and chronic conditions, and it becomes necessary to think in terms of liver function whenever the total body economy has been or is to be taxed. In fact, the liver should be regarded as the great dynamic chemical factory and regulatory center of the body which participates in an endless number of known and unknown metabolic processes.

Liver function depends largely on the integrity of the polygonal-cell, which is often damaged in hepatic disease. The other major components of the liver include: the Kupffer cells, which are a part of the reticuloendothelial system; the bile duct system; the vascular network, i.e., the hepatic artery and its branches, the portal system, the hepatic sinusoids, the hepatic veins and the hepatic lymphatic system, and the stroma, a connective tissue framework which plays an important role in the reaction of the liver to injury.

The liver has a double interconnecting blood vascular system. The hepatic artery supplies about 25 per cent of the blood entering the liver and is the chief source of oxygen. The portal vein contributes about 75 per cent of the blood to the liver and conveys the products of digestion, as well as insulin and other substances. The hepatic

sinusoids receive blood from both the arterial and the venous inflow systems and drain to the central veins, which convey blood to the hepatic veins, which in turn drain to the systemic circulation (inferior vena cava).

The bile ducts of the liver are in intimate relationship with the polygonal cells and serve as conduits for the passage of bile from the liver to the intestine. The bile canaliculi lie between the cords of hepatic cells and drain to the interlobular ducts, then to the left and right hepatic ducts, which form the common hepatic duct at the liver hilus. The common hepatic duct becomes the common bile duct where the cystic duct enters the duct system. The common bile duct terminates at the papilla of Vater in the second portion of the duodenum.

The liver has a remarkable degree of both functional reserve and regenerative capacity. This is illustrated by the fact that in the experimental animal about 80 per cent of the liver can be removed without severe functional impairment and, following this, regeneration will occur with restoration to normal size in six to eight weeks. Furthermore, even when serious liver damage is produced, all hepatic functions may not be disturbed to the same degree. Some hepatic functions may be greatly impaired while others remain normal or nearly normal. This has been called "dissociation" of functions. It is related to the type and degree of hepatic cell damage and varies according to time factors. It is possible that some functions which may be altered at one time, then become normal and others become disturbed. It should be apparent that hepatic functions are subject to great variation and must not be judged simply on the basis of function tests, but instead must be evaluated with respect to all available clinical and biochemical data.

Broadly speaking, the functions of the liver (Fig. 87) may be considered under three categories: metabolism, detoxification and excretion. Many materials enter the liver, where they are reduced to intermediate compounds, reconstituted, and stored or excreted in the original form or in new forms. The polygonal cell performs most of this metabolic work. Monosaccharides entering the liver are polymerized to glycogen and stored, or metabolized to the production of protein or fat, or formed into the polysaccharide heparin. Amino acids liberated in the intestine through digestive processes and transported to the liver are aggregated into new protein molecules which are required for maintenance and growth of tissues, or converted into

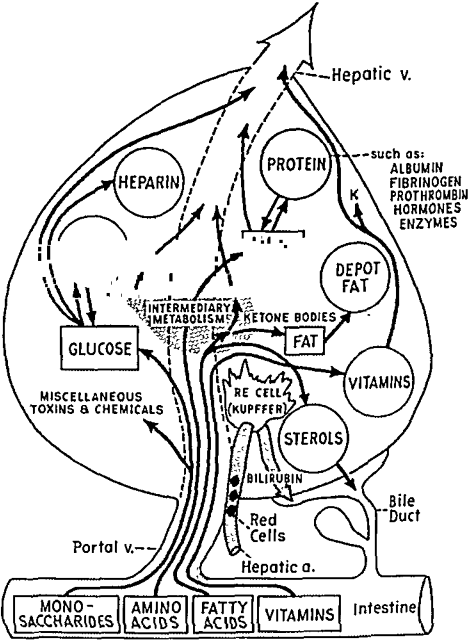

FIG. 87.—Schema of liver functions. Note the many functions of the polygonal cell, and the zone of intermediary metabolism, where much of the work of the liver is done. (Modified from Pfizer spectrum.)

other types of amino acids, or deaminized and converted into carbohydrate or fat. The amino groups (NH_2) that are split off are synthesized to urea in the liver. Fatty acids are also reshaped by the liver for storage or combustion. Liver fats are in dynamic equilibrium with body fats; and in the liver most fats are merely in transit, only a small portion being stored. Fats may be oxidized to ketone bodies and burned, converted into carbohydrate, used in the synthesis of cholesterol or phospholipid or stored.

The liver is the main reservoir of sugar in the body. Here, during absorption, glucose is converted to glycogen and stored. Glucose is delivered to the blood stream and to the tissues as needed between meals. Although many factors are involved, the liver plays a dominant role in the regulation of the blood sugar. Under conditions of liver injury, the hepatic cells are depleted of glycogen reserves and become increasingly susceptible to the damaging effects of various toxic agents (chloroform, bacterial toxins, anoxia, etc.). This is the basis for the time-honored practice of administering large amounts of carbohydrate to patients with liver disease.

The liver is concerned in the elaboration and maintenance of the plasma and tissue proteins. Albumin, fibrinogen and prothrombin are products of liver metabolism, while the globulins are formed outside the liver, particularly in the cells of the reticuloendothelial system. In serious hepatic disease the levels of the circulating proteins are often decreased. In health, some newly synthesized protein is stored in the liver, where it can be easily mobilized. Under conditions of malnutrition, these stores become depleted; and, as is the case when liver glycogen is depleted, the hepatic cells become more susceptible to the injurious effects of endogenous and exogenous toxins. On the other hand, when the liver contains increased amounts of fat, it also exhibits an increased susceptibility to the effects of hepatic toxins. Optimum protection to the liver from these adverse effects can be afforded by relatively high levels of carbohydrate and protein storage and low fat storage.

Vitamin metabolism and hepatic function are also interrelated. In liver disease with failure of bile production or secretion, poor intestinal absorption of the fat-soluble substances (vitamins A, D, E and K) occurs. In advanced liver disease, defects in intermediary metabolism can lead to faulty utilization or storage of other essential substances, especially the B complex and erythrocyte-maturing factors.

The liver is also concerned in the metabolism of minerals, such as iron, copper, manganese and zinc.

The liver forms and excretes bile continuously, and thus serves as one of the main excretory organs of the body. Bile is chiefly a mixture of water, bile pigment, bile salts and cholesterol. Bile pigment is formed from hemoglobin released from red blood cell breakdown. Hemoglobin is a specific protein, globin, to which heme, the pigment radical, is attached. Iron is essential for the respiratory properties of heme. The heme molecule is made up of iron and four pyrrole rings united by methene bridges (—CH). In some manner, the heme ring is broken at the alpha methene bridge by the reticuloendothelial cells. The result is a straight-chain iron-biliverdin-globin compound which has the characteristic color of the verdohemoglobins. This substance is rapidly reduced to a bilirubin-globin complex. Bilirubin is separated from its protein portion in the liver and excreted. After reaching the intestine, bilirubin is converted to urobilinogen through the action of bacteria. A whole family of urobilinogens and urobilins is formed, and these in large measure account for the normal brown color of the feces. Some urobilinogen is reabsorbed and returns, via the portal system, to the liver. Here some is transformed back to bilirubin and re-excreted, and a small amount enters the systemic circulation and is excreted by the kidneys.

The bile salts, sodium taurocholate and sodium glycocholate, are produced in the liver and secreted in the bile. They are formed by the conjugation of cholic and desoxycholic acid, derived from cholesterol, with taurine and glycine. Normally, the bile salts are reabsorbed from the intestine and resecreted. Thus the body conserves bile salts by means of an enterohepatic cycle. With either intrahepatic or extrahepatic blockage to bile flow, bile salts accumulate in the circulation. The pruritus of jaundice, which is most severe in extrahepatic obstruction, is believed to be due to excessive amounts of bile salts in the blood.

Normally, cholesterol and fatty acids are joined in the liver to form cholesterol esters, which constitute approximately 70–75 per cent of the total circulating cholesterol. The exact role of the liver in the metabolism and excretion of cholesterol and cholesterol esters is incompletely understood. In hepatic disease the levels of these substances are altered, most often in the direction of increased levels with obstruction of the bile passages and decreased levels in hepatic parenchymal disease; but the findings are quite variable.

JAUNDICE

Jaundice exists when there is retention of bilirubin in the blood. It is produced by three primary disturbances:

1. Excessive breakdown of red blood cells (hemolytic jaundice, acholuric jaundice, prehepatic jaundice)
2. Disturbances of the hepatic parenchyma (hepatocellular jaundice, retention jaundice, intrahepatic jaundice or medical jaundice)
3. Obstruction to bile outflow from the liver (obstructive jaundice, regurgitation jaundice, posthepatic or extrahepatic jaundice, or surgical jaundice)

The clinical and laboratory features of hepatocellular and obstructive jaundice are often quite similar. It is important, therefore, for the clinician to have a clear understanding of the basic mechanisms which produce these conditions, the methods available for differentiating them and the types of treatment required. It should be apparent that surgical treatment in the former is likely to be harmful, while delay incident to medical treatment in the latter may also be harmful.

Hemolytic jaundice usually poses no serious problems in diagnosis or treatment. In its uncomplicated form it is unassociated with disease of the liver or bile passages and will not be considered further in this discussion.

Hepatocellular jaundice results from primary injury to the liver cells. It is due to infections, hepatotoxic agents, nutritional deficiencies or metabolic disorders which lead to interference with the bilirubin transport mechanism, as well as other disturbances in hepatic cell function. It is not amenable to surgical treatment and is sometimes referred to as "medical jaundice."

Obstructive jaundice is usually due to the following conditions:

1. Common bile duct stone or stones (choledocholithiasis)
2. Malignant tumor obstructing the bile ducts (the tumor may be a primary cancer of the head of the pancreas, of the ampulla of Vater or of the gallbladder and bile ducts, or secondary metastatic cancer to the liver hilus or the liver parenchyma)
3. Stricture of the common bile duct or common hepatic duct, usually following duct injury during cholecystectomy
4. Constriction or compression of the common bile duct as a result of chronic pancreatitis
5. Congenital atresia or aplasia of the bile ducts in the newborn

When obstruction to the bile passages develops, there is inevitably some associated damage to the liver cells from interference with hepatic circulation, back pressure in the bile passages and bile stasis and infection, leading to hepatic cell injury and impaired function, with fibrosis and permanent damage if obstruction is prolonged. The degree of liver damage is directly related to the degree and duration of the obstruction to the bile flow. In the early stages of obstructive jaundice there may only be slight alterations in liver cells, and the liver function tests are usually normal.

The patient with jaundice poses at least three important questions: Is the jaundice of hemolytic, obstructive or nonobstructive origin? If the jaundice is of obstructive origin, what is the probable cause or causes? Is operative treatment indicated, and, if so, when should it be undertaken?

The differential diagnosis of jaundice can be established in the great majority of cases (about 80–85 per cent) on the basis of a careful analysis of the clinical history, physical examination and a few simple tests of the blood, urine and stool. In a small group of patients, repeated observations and more complicated tests of bile pigment metabolism and liver function are necessary; and in the remainder, usually the diagnosis cannot be established short of abdominal exploration.

Generally, jaundice does not call for emergency treatment and there is time to observe and study the patient. This does not mean, however, that prolonged or unreasonable delay should be permitted simply because an accurate anatomic and pathologic diagnosis is desirable if, meanwhile, the patient's condition is deteriorating and the chances for possible beneficial operative treatment are decreasing. In general, the period of observation and preoperative treatment in surgical jaundice should not exceed a week or ten days, and it is usually less than this.

History

In considering the diagnosis, careful assessment should be made of the patient's age, sex, body habitus, past health, exposure to infectious diseases, hepatotoxic agents, viral hepatitis, dietary intake, alcoholism, blood or plasma transfusions and chlorpromazine (Thorazine®) administration. The latter drug has been found to produce obstructive jaundice of the intrahepatic type, which physiologically

and pathologically closely simulates that caused by extrahepatic block.

The mode of onset of jaundice and its relationship to pain, fever, chills, malaise and asthenia, rapid weight loss, nausea and vomiting will in many instances give a clue to the underlying disturbance. Similarly, the course of the jaundice (increasing, constant, fluctuating or decreasing), especially if corroborated by someone other than the patient himself, may be an indication of the underlying mechanism of its production. There is also a correlation between the degree of the interference with bile flow and changes observed in the color of the urine and the stool. For example, commonly the patient will observe that the urine becomes dark and the stool becomes light colored shortly before the onset of jaundice. During the course of jaundice, and often associated with improvement in the patient's symptoms, the urine will become lighter and the stool will become more normal in appearance. Cyclic changes of this type over a period of days or weeks may be correlated with intermittent or partial extrahepatic obstruction, such as occurs with a "ball valve" obstruction due to common bile duct stone. On the other hand, obstruction of the common bile duct due to carcinoma of the head of the pancreas is more likely to produce rapid, complete and progressive or sustained obstruction to bile flow without significant amelioration or fluctuation in the intensity of the jaundice or the color of the urine or stool. During this period the symptoms may also be expected to become more severe and the patient's general condition to deteriorate. Another item of some diagnostic importance is the association of obstructive jaundice with persistent blood (usually occult) in the stool. In the absence of an obvious cause for melena and jaundice, the possibility of an ulcerating and obstructing tumor of the papilla or ampulla of Vater must be considered.

Severe and relatively intractable generalized pruritus favors the diagnosis of obstructive jaundice but, per se, does not suggest the underlying mechanism of its production. The cause of pruritus is unknown, although it is presumed to result from the retention of bile salts. It is significant that pruritus disappears almost immediately following the release of obstruction to the flow of bile.

Physical Examination

There are certain items in the general examination which are important in differential diagnosis. These include: the general ap-

pearance of the patient and the intensity of the jaundice (which can be correlated roughly with the serum bilirubin level); the signs of weight loss and nutritional depletion; the degree of anemia; the presence of vascular spiders, chiefly over the trunk, upper extremities and the shoulder girdle; the presence of dilated veins over the abdomen (caput medusae), which are collateral channels around the obstructed portal system (e.g., Laennec's cirrhosis); the size, consistency and nodularity of the liver; ascites; distended palpable gallbladder; splenomegaly; and the presence of other masses or enlargements which may suggest the existence of abdominal cancer.

X-ray Examination

The radiographic studies may be helpful, but negative findings do not preclude consideration of those conditions which are suggested by the clinical examination. For example, in cancer of the head of the pancreas the duodenal loop may be widened by the tumor, but the absence of this sign does not necessarily exclude this diagnosis. The presence of a persistent deformity in the second portion of the duodenum at the location of the papilla of Vater suggests the presence of a tumor, but the absence of this finding does not rule out such a tumor. Stones in the gallbladder and/or common bile duct may contain enough calcium to make them opaque to x-rays. If gallstones are seen, the presumptive diagnosis could be jaundice due to calculous obstruction of the common duct, but the absence of stones on the plain film does not rule out this diagnosis.

When the patient is jaundiced, the usual method of cholecystography is not helpful and may be harmful. There is usually insufficient excretion or concentration of iodine-containing media to produce a shadow on the x-ray film. Recently an iodine-containing preparation (Cholografin®), which may be given intravenously and which is rapidly excreted by the liver, has proved useful for demonstrating the bile ducts even in presence of incomplete or partial blockage If the serum bilirubin level does not exceed about 3.5 mg./100 ml., the duct system can often be opacified by this substance; but if the degree of retention exceeds this level, it is unlikely that the examination will be helpful. By the use of Cholografin,® one may, in questionable cases, establish the presence (or absence) of obstruction to the duct system, the amount of dilatation and possibly the cause of the obstruction.

A summary of the clinical data which may be helpful in the differential diagnosis of jaundice follows:

PAST HISTORY:

1. Previous episodes of jaundice (hemolytic crises) and jaundice in other members of the family suggest the possibility of congenital hemolytic icterus.

2. A previous history of gallbladder colic suggests stone (or stones) in the common bile duct.

3. A history of a previous operation on the biliary passages suggests the possibility of stricture of the common duct or a remaining common duct stone (or stones).

4. A history of recent administration of hepatotoxic drugs suggests hepatocellular jaundice.*

5. A history of a recent acute illness suggests infectious hepatitis.

6. A history of chronic illness and/or of alcoholism and malnutrition suggests hepatic cirrhosis.

7. A history of administration of blood, plasma or intravenous medication followed in about three months by jaundice suggests homologous serum hepatitis.

ONSET:

1. A sudden onset of right upper quadrant pain, with chills and fever, followed by jaundice suggests stones in the common duct.

2. A sudden onset of jaundice, chills and fever, associated with an enlarged and painful liver, suggests hepatitis.

3. A slow onset of painless jaundice suggests obstruction due to cancer.

COURSE:

1. Jaundice appearing rapidly and then leveling off suggests intrahepatic disease.

2. Fluctuating or recurrent jaundice suggests common duct stones.

3. Progressive jaundice with light stools and dark urine suggests neoplastic disease.

PAIN:

1. Heavy, dragging upper abdominal pain suggests hepatic disease.

2. Sharp, colicky pain suggests common duct stones.

3. Severe deep, boring back pain, or *lack* of pain may suggest cancer of the pancreas.

DIGESTIVE DISTURBANCES:

1. A history of gallbladder symptoms over a period of years suggests the possibility of common duct stones.

2. Anorexia, rapid weight loss, and asthenia suggest malignant obstruction.

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*Unlike the jaundice resulting from most hepatotoxic drugs, chlorpromazine (Thorazine®) sometimes produces the clinical and laboratory picture of obstructive jaundice by virtue of intrahepatic obstruction.

3. A history of an inadequate food intake and alcoholism suggests the possibility of cirrhosis.

PRURITUS:

Variable and of little diagnostic value; but if severe, favors obstruction, either benign or malignant.

AGE AND SEX:

1. Jaundice in early life suggests a hemolytic or intrahepatic origin.

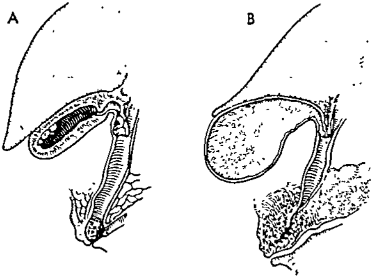

FIG. 88.—Courvoisier's rule in obstructive jaundice: *A*, when obstructive jaundice is due to stone in the common bile duct, the gallbladder tends to be small, contracted and not palpable, *B*, when obstructive jaundice is due to cancer of the head of the pancreas, the gallbladder tends to be large, distended and palpable. These findings are explained on the basis of pre-existing scarring and lack of distensibility due to chronic inflammation in cholecystitis with cholelithiasis, which is usually associated with common duct stone, and absence of scarring with normal distensibility of the gallbladder in neoplastic obstruction.

2. In later life, especially in the male, cancer of the pancreas and cirrhosis are common.

3. In later life, especially in the female, gallbladder disease and common duct stones are common.

SPIDER ANGIOMATA:

Favor parenchymal liver disease but are not diagnostic.

LIVER ENLARGEMENT:

1. If early in the course of jaundice, suggests intrahepatic disease.
2. If late, suggests extrahepatic disease.
3. A smooth, soft, tender liver suggests hepatitis.
4. A firm, nodular liver border suggests cirrhosis or metastatic cancer.

SPLENIC ENLARGEMENT:

1. Suggests hemolytic jaundice.

2. Consider congestive splenomegaly secondary to cirrhosis if other signs of hepatic disease are noted.

GALLBLADDER ENLARGEMENT:

1. If distended and palpable, favors neoplastic obstruction of the common duct (Courvoisier's rule, Fig. 88).

2. If gallbladder is not palpable, no diagnostic implication; but odds would favor calculous obstruction (ball valve) or parenchymal liver disease rather than malignant obstruction.

ASCITES:

1. Favors the diagnosis of cirrhosis or metastatic cancer to the liver or peritoneum.

2. Absence of ascites favors diagnosis of benign extrahepatic obstruction if jaundice is of long standing.

LIVER FUNCTION TESTS

There are many tests available for the evaluation of various liver functions. Obviously, no single test can be expected to indicate the host of activities which take place in this complex organ. Furthermore, the dynamic quality of liver metabolism is such that changes in the direction of either impairment or improvement may occur rapidly. Thus it is necessary to secure several tests of liver function, when they are indicated, and to perform them serially if necessary. The tremendous reserve and regenerative capacity of the liver and the tendency to dissociation of functions, already mentioned, points up the need for careful evaluation when the physician is faced with the problem of a chronically ill patient who presents normal laboratory findings. It is possible for the liver to be "sick" despite negative laboratory findings.

The liver function tests (Table 18) can be used to detect and measure various types of hepatic insufficiency, to estimate residual damage after recovery, to follow the trend of the disease, to evaluate the preoperative surgical risk, to aid in the selection and timing of surgical procedures and to assist in differentiating the type of jaundice. Because some grouping of laboratory procedures is necessary for orientation, these tests may be considered in two categories: excretion and metabolism. Since the detoxifying functions of the liver are actually metabolic functions, they may be combined under one heading.

EXCRETORY FUNCTIONS

SERUM BILIRUBIN.—As previously stated, the breakdown of red blood cells in the reticuloendothelial system yields a bilirubin-globin

TABLE 18.—Liver Function Tests ‡

TEST	NORMAL VALUES	PARENCHYMAL JAUNDICE	EXTRAHEPATIC OBSTRUCTIVE JAUNDICE	HEMOLYTIC JAUNDICE
ICTERUS INDEX	2 - 6	INCREASED 6-150	MARKED INCREASE 50-200	MILD INCREASE 6-75
[illegible]	[illegible]	[illegible]	[illegible]	[illegible]
[illegible] (QUALITATIVE)	[illegible]	[illegible]	[illegible]	[illegible]
TOTAL PROTEIN	6-8 GR/100 CC	DECREASED	* NORMAL	NORMAL
ALBUMIN/GLOBULIN RATIO	1.25-3.5	DECREASED	* NORMAL	NORMAL
TOTAL CHOLESTEROL	150-270 MG/100 CC	NORMAL/DECREASED †	INCREASED	DECREASED
CHOLESTEROL ESTER %	60-80 %	DECREASED	* NORMAL	NORMAL
ALKALINE PHOSPHATASE	1-4 BODANSKY UNITS	NORMAL OR MILD † INCREASE 4-15 B.U.	MARKED INCREASE 15-60 B.U.	NORMAL
CEPHALIN FLOCCULATION	0-1+	INCREASED 3-4+	* NORMAL 0-1+	NORMAL 0-1+
THYMOL TURBIDITY	1-4 UNITS	INCREASED	* NORMAL	NORMAL
ZINC SULFATE TURBIDITY	2-12 UNITS	INCREASED	* NORMAL	NORMAL
GALACTOSE TOLERANCE	0-3 GRAMS	INCREASED	* NORMAL	NORMAL
[illegible]	[illegible]	[illegible]	[illegible]	[illegible]
DUODENAL DRAINAGE	[illegible]	[illegible]	[illegible]	[illegible]
[illegible]	[illegible]	[illegible]	[illegible]	[illegible]

† IN SEVERE PARENCHYMAL JAUNDICE (INTRAHEPATIC OBSTRUCTION), THE BILIRUBIN AND UROBILINOGEN RESULTS AND RARELY THE OTHER FUNCTION TESTS MAY RESEMBLE THOSE FOUND IN EXTRAHEPATIC OBSTRUCTION.

‡ Courtesy of Dr. Murray Franklin.

complex which is carried to the liver, where the globin is split off and free bilirubin is excreted in the bile. This is a greatly oversimplified statement of the changes that occur.

Van den Bergh's *original conception of the different reacting portions of bilirubin* is still useful. He recognized two types of reactions occurring in different types of body fluids. When the diazo-reagent was added to bile or to the serum of patients with obstructive jaundice, a reaction occurred promptly. However, when this reagent was added to hemorrhagic fluid from body cavities or to blood serum from patients with hemolytic jaundice, a reaction was *slow to develop*. He noted that *if alcohol was added, the reaction became maximal* almost the same moment. The direct reaction is that which occurs promptly after the addition of the diazo-reagent alone; the indirect reaction is that which occurs after the addition of diazo-reagent and alcohol. The concept that there are two types of bilirubin—one "free" and yielding a prompt or direct reaction with diazo-reagent, the other bound to protein and giving a *delayed or indirect response to diazo-reagent—was based on these observations.*

The terms *direct* and *indirect* van den Bergh reaction fell into discard when it was determined that one could not, by this means, differentiate obstructive from hepatocellular disease, since practically all sera from jaundiced patients contain some of both fractions. In spite of this, the reaction still has considerable usefulness. The original *procedure has been modified by arbitrarily measuring the direct reaction* at one minute and the total at the end of fifteen to thirty minutes. The difference between the direct reacting fraction and the total bilirubin is the indirect fraction. The upper limit of normal is: 0.25 mg./100 ml. for one minute bilirubin and 1.0 mg. for total bilirubin.

The value of the bilirubin partition lies in the significance of the *one minute portion. Frequently in mild* or early hepatic disease the total bilirubin level will be *normal*, while the *one minute bilirubin is* abnormally elevated. The one minute bilirubin is of particular value in hepatitis in both the early and the convalescent stages and in mild cirrhosis. Usually, in uncomplicated hemolytic icterus the preponderance of elevated bilirubin is in the indirect fraction.

Icterus Index.—The ordinary method for determining the icterus index consists in comparing the *patient's serum to a standard solution* of 1:10,000 potassium dichromate, the color intensity of which is taken as unity. Other factors which add color to the serum during the tests will preclude accurate readings. The most common causes of error

are carotenemia, hemoglobinemia, lipemia and the presence of pyrrole compounds. Although the test is satisfactory for most clinical purposes, chemical determination of serum bilirubin is usually preferred. The normal values are 2–6 units.

BROMSULPHALEIN.—Bromsulphalein (BSP) retention is a most useful test of hepatic function in nonjaundiced patients and is of particular value in detecting marginal degrees of hepatic dysfunction. This substance is removed from the blood by the liver and excreted in the bile. Recent work indicates that BSP is picked up by the parenchymal cells, stored for a variable length of time and then excreted. The Kupffer cells probably play no part in BSP removal or excretion. The test is performed by introducing intravenously 5 mg. of the dye/kg. of body weight and removing a blood sample at forty-five minutes for the colorimetric determination. A value greater than 5 per cent retention is abnormal.

In hepatic disease, BSP removal is impaired by cellular failure and/or decreased hepatic blood flow. Reduced hepatic blood flow decreases the rate of BSP removal regardless of the functional state of the liver cells. In cardiac failure, abnormal levels of BSP retention may occur simply because decreased hepatic blood flow exists. Emergency BSP determinations have been advocated as an aid in the differential diagnosis of acute gastrointestinal hemorrhage from esophageal varices associated with cirrhosis and peptic ulcer. Although the test is of value in this connection, one must remember that shock from blood loss may be responsible for false positive reactions.

ALKALINE PHOSPHATASE.—"Phosphatase" is a term applied to one or more enzymes hydrolyzing phosphoric acid ester substrates. The several methods employed in its determination are all similar in that they measure the phosphate set free as inorganic phosphate when the enzyme present in the blood is permitted to act on a phosphoric ester substrate under standardized conditions. The Bodansky method is commonly used in this country. The normal range is 1–4 Bodansky units.

The exact nature of circulating phosphatase is unknown. The enzyme is produced in bone and in other tissues, including the liver. Rapidly proliferating cells, such as certain carcinoma cells or regenerating liver cells, are particularly prolific in their production of this substance. Biliary excretion of phosphatase is well established, and it has been shown that all of the rise associated with obstruction of the biliary passages is not due to delayed excretion per se. The highest

blood levels of alkaline phosphatase are found in obstructive jaundice; however, considerable elevation occurs in the absence of jaundice in such conditions as bone tumors and metastatic carcinoma of the liver. In experimental animals, ligation of a small segment of bile ducts will produce elevated phosphatase values without bilirubin retention. Thus it appears that there is a mechanism present for the selective retention of phosphatase. Proliferating bile duct epithelium is especially prone to produce abnormal results, and high levels are often encountered in biliary cirrhosis.

It has been observed that, when the alkaline phosphatase is above 15 Bodansky units and jaundice is deepening, obstruction is likely; whereas, if the level is below 10 Bodansky units, diffuse damage of the hepatic parenchyma is probable. The diagnostic value of the test is limited by the fact that moderate rises may occur in jaundice due to either obstruction or hepatitis. However, normal values are unusual in obstructive jaundice.

Urine and Fecal Bile and Urobilinogen.—The tests for bile and urobilinogen in the stool and in the feces are of considerable clinical importance. They are simple and easy to perform; the reagents are stable; and much useful information can be obtained through their routine use.

Under normal conditions (Fig. 89, *A*) 40–280 mg. of urobilinogen appears in the feces per day and 0.5–3 mg. in the urine. Alterations in the excretion of these substances can be detected by simple qualitative methods or exact quantitative tests.

Patients with uncomplicated hemolytic jaundice (Fig. 89, *B*) form bilirubin in abnormal amounts, but hepatic function is generally normal. There is increased urobilinogen in the stool and in the urine. Bilirubin does not appear in the urine. Patients with jaundice due to hepatocellular disease (Fig. 89, *C*) have increased amounts of urobilinogen and bilirubin in the urine and some urobilinogen in the stool. Patients with jaundice due to complete bile duct obstruction, e.g., cancer of the head of the pancreas (Fig. 89, *D*), have large amounts of bilirubin in the urine but no urobilinogen in the urine or feces. With intermittent bile duct obstruction (e.g., "ball valve" common duct stone), intermittent increases in bilirubin in the urine and urobilinogen in the urine and stool are observed if daily determinations are made.

Simple methods have been devised for the detection of bilirubin in the urine, and kits for this purpose can be obtained. A color reaction

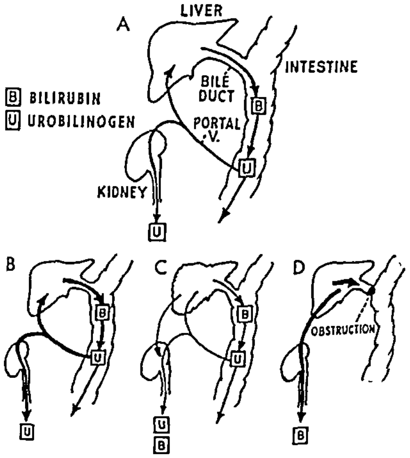

FIG. 89.—Bile pigment excretion in normal and jaundiced patients. *A*, normal. [illegible] the urine. *D*, obstructive jaundice (complete). No urobilinogen is excreted in the stool or urine. Bilirubin is excreted in the urine.

is produced which can be graded from a trace to 4+, and a relatively accurate semiquantitative appraisal obtained.

The standard method for the semiquantitative determination of urine urobilinogen is the dilution method of Wallace and Diamond. A more simple procedure is that of grading Ehrlich's test on a trace to 4+ basis. With practice, the color intensity can be graded closely enough to make the test useful clinically.

The feces are tested for urobilinogen in a similar manner. Since urobilin is also present in normal feces, the relative amount of this substance is determined by detecting the green fluorescence imparted

to fecal filtrates when a saturated alcoholic solution of zinc acetate is added (Schlesinger test).

Metabolic Functions

The tests of the metabolic functions of the liver may be considered as they pertain to carbohydrate, fat and protein.

Glucose metabolism remains relatively unaltered in liver diseases except in such conditions as severe cirrhosis and massive hepatic necrosis. For this reason, specific tests for glucose metabolism have not proved of practical diagnostic value.

Galactose Tolerance.—Galactose is a sugar which is metabolized solely in the liver, where it is converted to glycogen. Any impairment of this reaction will cause abnormal amounts of galactose to appear in the blood and urine. The most commonly used test is performed by giving 100 ml. of a 25 per cent solution of galactose intravenously and analyzing blood samples for galactose at sixty, seventy-five and ninety minutes. Normally the liver can metabolize about 25 Gm. of galactose in one hour, and any remaining in the blood after this period constitutes a positive test.

It is generally believed that the galactose tolerance test is suitable for estimating the carbohydrate metabolism of the liver. Approximately 80 per cent of the patients with obstructive jaundice have normal tolerance tests, while only 30 per cent of patients with hepatocellular disease have normal tests. These results are no better than those obtained with other function tests; therefore, the test is not widely used.

Cholesterol and Cholesterol Esters.—The most important measure of liver fat metabolism is the blood cholesterol. Although the exact role of the liver in cholesterol metabolism remains unknown, it has been observed that alterations in the levels of free and esterified cholesterol occur in liver disease and obstructive jaundice. Cholesterol is normally present in bile, and the increased blood levels which occur in association with obstruction to bile outflow are understandable; but the decreased levels of cholesterol which occur with severe hepatic damage are more difficult to explain. Because factors beyond the liver are known to influence cholesterol metabolism, it is perhaps better to regard these abnormalities in cholesterol as indications of liver dysfunction rather than as tests of liver function. In obstruction, the rise in total cholesterol level tends to parallel that of bilirubin; while in severe parenchymal disease the cholesterol tends to fall as the bilirubin rises.

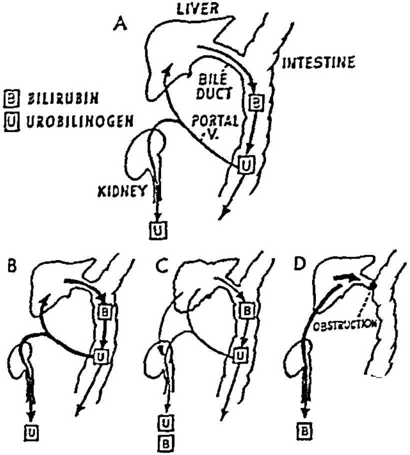

FIG. 69.—Bile pigment excretion in normal and jaundiced patients. *A*, normal. Urobilinogen (and urobilin) is excreted in the stool. Some urobilinogen is reabsorbed from the gut and excreted in the urine. *B*, hemolytic jaundice. Increased amounts of urobilinogen are excreted in the stool and urine. *C*, hepatocellular jaundice. Urobilinogen is excreted in the stool and urine. Bilirubin is also excreted in the urine. *D*, obstructive jaundice (complete). No urobilinogen is excreted in the stool or urine. Bilirubin is excreted in the urine.

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The normal cholesterol level is subject to great individual variation but is relatively constant for any one patient. Thus, serial determinations are often important. The generally accepted normal range is 150–250 mg./100 ml.

In the past, the regulation of cholesterol esters has been ascribed entirely to the liver, but recent studies have shown that esterification occurs in the blood as well as in the liver. The cholesterol ester level is rather constant at 70–75 per cent of the total cholesterol. In parenchymal liver disease, there is generally a decline in both the cholesterol and cholesterol esters; however, the decrease in the ester fraction is out of proportion to that of the total cholesterol. Considerable importance has been attached to a decreasing ester fraction, and a large degree of fall has been considered a sign of poor prognosis. Nevertheless, it was noted that in some cases of pure obstructive jaundice a rapid fall in cholesterol esters occurred without any other evidence of diffuse liver disease. It has also been shown that there is an inverse relationship between the serum bilirubin and the cholesterol ester levels, regardless of the cause of the bilirubinemia. Thus, one must be careful when interpreting low ester values in jaundiced patients. As with the serum alkaline phosphatase, there may be a rapid rise in cholesterol and cholesterol ester levels during periods of liver cell regeneration. Generally, the serum bilirubin tends to fall at this time.

The determinations of other fractions of the blood lipids are more complicated and of little practical value in the management of patients with liver disease.

Hippuric Acid.—Benzoic acid is conjugated with glycine in the liver to form hippuric acid, which is excreted in the urine. The main site of glycine synthesis is the liver; therefore, a deficiency of glycine resulting from liver damage will decrease the formation of hippuric acid. The usual test is to administer a solution of 1.77 Gm. of sodium benzoate intravenously and collect all urine voided during the first hour. The minimal output of hippuric acid is 0.7 Gm. Values below this level indicate diffuse cellular damage, provided adequate preliminary intake of carbohydrate and protein has occurred and there is good renal function. Comparative studies have shown that the test is somewhat more sensitive than the galactose tolerance and cholesterol partition but less sensitive than the urine urobilinogen and the BSP determinations.

Plasma Proteins.—It is well established that the liver is the site of albumin formation. It is not surprising, therefore, that serum

albumin level is decreased in hepatocellular disease. Hypoalbuminemia is the most consistent alteration of the serum proteins found in patients with chronic advanced liver disease. The present concept is that the decrease in albumin is due to failure of liver synthesis rather than to failure of protein intake. The normal values are: serum albumin, 3.2–4.2 Gm./100 ml.; serum globulin, 2.8–3.8 Gm.; and total serum proteins, 6.0–8.0 Gm.

Less constant is the rise in total serum globulin and the reversal of the albumin-globulin ratio. The globulins are, for the most part, synthesized outside the liver, and the mechanism of their reciprocal increase with albumin is unknown.

It is fair to say that, if the serum proteins are abnormal, the probabilities are that severe hepatic dysfunction exists; but it must be remembered that normal levels may be found in the presence of liver disease.

PROTHROMBIN.—Prothrombin formation is directly related to vitamin K absorption and to the capacity of the liver to utilize this vitamin in the formation of prothrombin. The absence of bile salts from the intestine, with failure of absorption of vitamin K, or severe hepatic cell damage with failure of prothrombin synthesis, causes a reduction in plasma prothrombin and a bleeding tendency. Hypoprothrombinemia can be detected by the use of one of several methods for estimating prothrombin. If low values are found, synthetic vitamin K can be administered; and the rate and magnitude of the response to vitamin K, as measured by serial prothrombin tests, will be an indication of this liver function (prothrombin response test). In obstructive jaundice a prompt rise in prothrombin will generally follow, while in hepatocellular jaundice the response will be roughly proportional to the extent of cellular damage. From a practical viewpoint, however, the one-stage prothrombin test is not a very sensitive indicator of hepatic function; and the test is further limited by the laboratory accuracy in performing it. In surgical patients the value of the prothrombin determination lies largely in the detection of a hemorrhagic tendency and in the control of Dicumarol® treatment for thromboembolic disease.

FLOCCULATION TESTS.—The seroflocculation tests are not strictly tests of liver function. They indicate alterations in serum proteins and lipid. Since the liver controls the levels of these substances, they reflect alterations in liver function.

Cephalin-Cholesterol Flocculation.—Hanger (1939) noted that

the sera of subjects with hepatic parenchymal damage flocculated a colloidal suspension of cephalin-cholesterol complex. It has been found that the chief factor responsible for this reaction is a labile alteration in the albumin factor. This factor, which is present in normal sera and apparently elaborated by the liver, prevents flocculation of the colloidal cephalin-cholesterol suspension. In liver disease the stabilizing substance is decreased and flocculation occurs. Positive results are reported as 1 + to 4 +, depending on the amount of flocculation present. The forty-eight hour reading is more dependable than the twenty-four hour, and all sera should be observed for this longer period before a final decision regarding positivity or negativity is made. In most laboratories a 2 + or greater reaction in forty-eight hours is considered positive. Values less than this are frequently encountered in febrile and other presumably nonhepatic disorders.

The cephalin-cholesterol flocculation is one of the most sensitive tests and probably the most useful of all the currently employed flocculation reactions. It is nearly always positive in early hepatitis and in active cirrhosis. A persistent, highly positive reaction is an omen of poor prognosis in cirrhosis and chronic hepatitis. The test is generally negative in obstructive jaundice until cholangitis and/or severe cell damage supervene.

Thymol Turbidity.—Thymol and other phenolic reagents precipitate globulin from certain pathologic sera, notably that from patients with liver disease. The precipitate consists of a globulin-thymol-lipid complex. Positive thymol flocculation occurs in the presence of elevated gamma or beta globulin, but there is no correlation with total globulin level. In addition, phospholipid must be present, and if there is a lowered albumin level, flocculation occurs more rapidly.

In most laboratories, 5 units or greater is considered a positive test. There is a high incidence of abnormal values (about 90 per cent) in parenchymal liver disease. A positive thymol test may be considered suggestive of liver disease. A strongly positive value implies significant hepatic involvement. Under these conditions, other confirmatory liver function tests should be positive.

Zinc Sulphate Flocculation.—Patients with liver disease frequently have high levels of serum gamma globulin. Weak solutions of zinc sulphate precipitate this substance, and this reaction has been utilized as a test of liver damage.

The generally accepted normal value is 4–12 units. The reaction is positive in diseases with an abnormal content of serum gamma

globulin, and in this respect it parallels the erythrocyte sedimentation rate. *The reaction is, therefore, nonspecific for liver disease and is less reliable than the cephalin flocculation or the thymol turbidity.* Despite its nonspecificity, the zinc sulphate flocculation is a valuable test of hepatic dysfunction, *especially if used in conjunction with the* cephalin-cholesterol reaction.

TREATMENT OF THE PATIENT WITH IMPAIRED LIVER FUNCTION

When the patient has been fortified with adequate amounts of *carbohydrates, proteins and vitamins,* he is better able to withstand the stresses of anesthesia and operation. On the other hand, when the patient is "nutritionally bankrupt," he is most susceptible to shock, *infection, edema, delayed wound healing and other complications.* In large measure, the adequacy of the metabolic response is dependent on the functional capacity of the liver.

Under conditions of impaired liver function, much can be done to bolster the liver function by proper dietary management. Natural foods give the best results. When the oral route is unavailable, artificial feedings and/or parenteral solutions must be used. At present, however, complete nutritional balance cannot be established or maintained through the parenteral route alone.

The therapeutic regimen should include the following items:

Diet—*The diet should have a high protein, high carbohydrate* and moderate fat content. An intake of about 3,000 calories is recommended, consisting of approximately 350 Gm. of carbohydrate, 125–175 Gm. of protein and 70–100 Gm. of fat. Butter, cream and eggs should not be excluded. The diet should be palatable and attractively served.

Vitamins.—Vitamins should be supplied in adequate amounts (parenteral routes if necessary), including A, B complex, D and K.

Antibiotics.—The broad-spectrum drugs, given by mouth if possible, are indicated, especially if there is evidence of sepsis. The tetracycline drugs are excreted in high concentration in bile.

Blood Transfusion.—The blood volume, hemoglobin and serum protein levels should be restored to normal or near normal. Fluid and electrolyte imbalances must be corrected.

Operative Intervention.—The type and time of surgical treatment depends on existing conditions.

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PORTAL HYPERTENSION

The condition in which the venous pressure in the portal system is elevated above normal levels (about 150 mm. H_2O) as a result of obstruction located either outside (extrahepatic) or inside (intrahepatic) the liver is called "portal hypertension" (Fig. 90). Most

FIG.
tension re
of massiv
rarily by tamponade with the Sengstaken-Blakemore tube. Portacaval anastomosis was planned, but the patient refused operation and later died of hemorrhage and liver failure.

commonly, portal hypertension is caused by cirrhosis of the liver. Less often it is caused by thrombosis or compression (tumors, parasitic disease, etc.) of the portal or splenic veins. The important clinical features are: enlargement of the spleen (congestive splenomegaly); the blood changes of hypersplenism (e.g., anemia, leukopenia, throm-

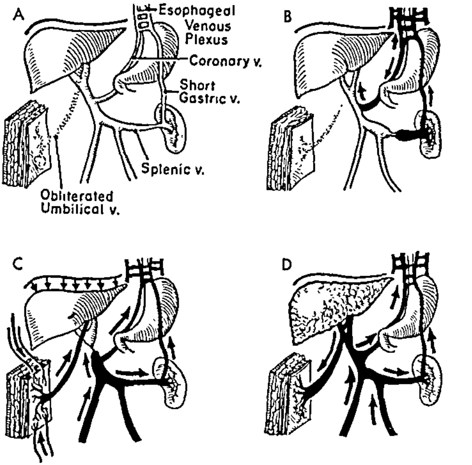

FIG. 90.—Portal vein obstruction with portal hypertension and esophageal varices. The collateral circulation in extrahepatic and intrahepatic obstruction are shown. A single vein is used to represent a plexus of veins. *A*, normal portal vein relationships. Note the coronary vein of the stomach and short gastric veins, both of which communicate with the esophageal plexus. Also note the obliterated umbilical veins. *B*, isolated splenic vein obstruction (thrombosis, cancer, parasitic disease,

veins, coronary vein and short gastric veins Portacaval, or splenectomy and splenorenal shunt should be considered for bleeding from varices. (From Learmonth, J R, and MacPherson, A. I. S, Surgery of portal hypertension, Lancet 2:882, 1948.)

designed to shunt blood from the portal to systemic veins and thus to effect a reduction in portal vein pressure. The results from these operations appear to be better than those from other procedures.

Ligation of the Hepatic Artery.—If the arterial inflow to the liver is decreased, the competition between arterial and portal inflow is said to be eliminated and the portal pressure falls. This procedure may result in hepatic necrosis and death from liver failure and is not recommended.

Splenectomy.—Although portal pressure is decreased temporarily by removal of the spleen, the symptoms of portal hypertension soon return. Simple splenectomy is therefore inadvisable. Splenectomy combined with immediate splenorenal anastomosis is recommended.

Patients with portal hypertension frequently have diffuse liver disease and are poor candidates for immediate surgical treatment. They must be carefully selected and prepared for operation. Shunting operations are difficult, postoperative complications are common, and the liver disease may progress despite the fact that a satisfactory lowering of portal pressure has been achieved. The treatment of portal hypertension is difficult under the best of circumstances.

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bopenia); esophageal varices (Fig. 91), recurrent bleeding from ruptured varices; and usually signs of cirrhosis of the liver. Associated findings may include: ascites, hemorrhoids, dilated abdominal veins (collateral channels), malnutrition and some degree of liver failure. The esophageal varices represent dilated collateral veins between the portal and caval systems. The varices tend to rupture and bleed massively. Portal hypertension is an exceedingly complex problem, and treatment is difficult. The extrahepatic type of portal hypertension is most amenable to surgical correction but is the least common type.

Recurrent hemorrhage from esophageal varices is a serious complication and demands consideration of operative treatment. At the present time, two general approaches to these problems are available: (1) direct measures to control or remove the varices and (2) indirect measures designed to reduce the portal pressure.

Direct Measures for Treatment of Bleeding Esophageal Varices

Balloon Tamponade (Sengstaken-Blakemore tube).—Temporary emergency control of bleeding is provided by compression of the varices with an inflatable balloon.

Injection of Varices.—By means of sclerosing solutions through an esophagoscope, temporary control of bleeding may be secured. Recurrent hemorhage is usual.

Resection of a Segment.—Following resection of a segment of the varicosed esophagus, an intrathoracic esophagogastric anastomosis is established Results are often unsatisfactory.

"Reefing."—By "reefing" or suture of the varicosities through the opened esophagus by transthoracic operation, obliteration of the varicosities and immediate control of bleeding may be accomplished, but recurrent hemorrhage is likely. The portal hypertension is not relieved.

Indirect Measures for Bleeding Esophageal Varices

Portacaval or Splenorenal Shunt.—The portal vein is anastomosed to the inferior vena cava (end to side or side to side), or the spleen is removed and the central end of the splenic vein is joined to the side of the renal vein. The kidney is not removed. These procedures are

CHAPTER 19

The Pancreas

THE PANCREAS is a retroperitoneal gland lying in a *transverse position* at about the level of the first lumbar vertebra. It extends from the convexity of the duodenum to the hilum of the spleen and roughly forms a horizontal inverted letter J. The hooked part of *the J, or head, lies within the duodenal sweep and over the right renal* vessels and the inferior vena cava. It is traversed by the common bile duct, as well as by the pancreatic ducts, in their course to the duodenum. In addition to the head, *the pancreas is composed of the* narrowed neck, which overlies the superior mesenteric vessels and portal vein; the body, which lies on the aorta; and the tail, which passes anterior to the left kidney. The mesentery of the transverse colon arises from the anterior border of the body and tail of the pancreas. Most of the pancreas lies above the mesentery and in the posterior wall of the lesser peritoneal cavity. Anteriorly, the pancreas lies on a level with the stomach, the transverse portion of the colon and the omentum.

The pancreas is a yellowish white, lobulated, soft structure which is similar in many respects to the parotid and submaxillary glands. It is liberally supplied with blood vessels, lymphatics and nerves. In keeping with its foregut origin, its blood supply is derived chiefly from the celiac artery and its nerve supply from the vagus and splanchnics (T5 to T9).

The duct system consists of two parts: (*a*) the main pancreatic duct (Wirsung), which begins distally in the tail and extends the length of the gland to enter either the ampulla of Vater, the papilla of Vater or the duodenum independently; and (*b*) the accessory pancreatic duct (Santorini), which drains the upper portion of the head and enters the duodenum independently, proximal to the papilla of

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clinical manifestations of acute pancreatitis, chronic pancreatitis, cancer of the pancreas or functioning islet cell tumors are prominent. No doubt there are many patients with early or occult pancreatic disease who are treated for other conditions, including the psychoneuroses, or are dismissed without treatment. Some of these errors on the part of physicians can be attributed to a lack of understanding of the disorders of the pancreas and the measures available for their detection and treatment. It must be admitted, however, that even under the best of circumstances the recognition and treatment of pancreatic disease is often difficult.

There are certain clinical findings which point to disturbances of the pancreas and which should suggest to the physician the need for investigating this organ by all the means at his disposal, including, in selected cases, surgical exploration. The important signs of pancreatic disease follow:

1. There is upper abdominal (foregut) pain, which may radiate to either side of the abdomen and/or straight through to the back. The pain can be intermittent or constant, mild or very severe. Often it is aggravated at night when the patient is supine, and is relieved when sitting up, bending forward or pressing on the abdomen. In contradistinction to the story in peptic ulcer, the pain is worse after eating.

2. Weight loss tends to be rapid and progressive in pancreatic diseases associated with duct obstruction and/or parenchymal destruction. Loss of weight in pancreatic cancer is outstanding and often antedates the appearance of other signs.

3. Indigestion, loss of appetite, fulness after meals, heartburn, belching, nausea, distaste for food or a peculiar taste in the mouth should suggest the possibility of pancreatic disease if the symptoms cannot otherwise be accounted for.

4. Disturbed bowel function with frequent, loose, bulky, light-colored and often foul-smelling stools results from poor digestion of ingested fat and protein.

5. Jaundice may be present, but the significance of this sign as an indication of pancreatic disease has been overemphasized. While it is true that obstructive jaundice can result from edema, inflammation or neoplastic infiltration of the head of the pancreas, jaundice does not commonly appear with lesions of the body or tail. The absence of jaundice, therefore, does not rule out the possibility of pancreatic disease; nor does the presence of obstructive jaundice necessarily

Vater. It also communicates with the main pancreatic duct within the gland.

Physiologically the pancreas is a dual organ. The exocrine portion, which constitutes the bulk of its substance, is concerned with the production and delivery of digestive enzymes to the intestine. The endocrine portion, the islands of Langerhans, is the source of hormones concerned with the metabolism of carbohydrates (insulin and glucagon).

The external secretion of the pancreas is a colorless, slightly mucoid enzyme-rich alkaline fluid amounting to about 400–600 ml. daily. Some pancreatic fluid is secreted continuously, but its volume and enzyme concentration vary according to neurogenic and hormonal stimulation. The neurogenic stimulation of the pancreas is provoked by eating and results in the secretion of juice high in enzyme content; the hormonal stimulation, which occurs through secretin liberated from the duodenum in response to food and acid, results in the secretion of large amounts of juice high in bicarbonate and low in enzymes.

The main pancreatic ferments—trypsinogen, amylase and lipase—are specific substances concerned with the digestion of protein, starches and fats, respectively. Other enzymes, including chymotrypsin, carboxypeptidase, lactase, maltase and sucrase, are also present but in lower concentration. Trypsinogen normally is activated to trypsin by enterokinase and by trypsin itself and calcium ions in the intestine. Pancreatic lipase splits neutral fats into fatty acids and glycerol and aids in the emulsification of liquid fats. Lipase in combination with bile salts accelerates the digestion of fats.

The internal secretions of the pancreas are elaborated by the islet cells and discharged directly into the blood stream. The islands of Langerhans, formed by a process of sequestration and growth of highly vascularized cells of the pancreatic duct system, are most numerous in the tail and body. Insulin and glucagon are produced by the beta and alpha cells, respectively, and participate in the complex processes of carbohydrate metabolism. The many interesting facets of this subject cannot be detailed here.

CLINICAL CONSIDERATIONS

Disorders of the pancreas are probably much more common than generally realized. Usually the diagnosis is made only when the

disease beyond weight loss, abdominal tenderness, occasionally a distended gallbladder and rarely a palpable epigastric mass. If it is borne in mind that the pancreas lies deep within the abdomen, completely covered by other structures, the limitations to palpation will be better understood. Except when the patient is very thin, the abdominal muscles well relaxed and the disease process extensive, palpation of the pancreas will be unrevealing. The finding of a distended gallbladder in a patient with obstructive jaundice (Courvoisier's rule) is, however, suggestive of pancreatic cancer (Fig. 92) but does not necessarily indicate advanced cancer.

In suspected acute pancreatitis, the laboratory finding of an elevation of the serum amylase or the serum lipase is diagnostic. In chronic diseases of the pancreas the blood enzyme studies are normal and of no value, but pancreatic secretion tests are often helpful.

For the pancreatic secretion test, pancreatic fluid is collected from the duodenum undiluted with gastric juice, using a double-lumened tube, one lumen of which allows for removal of gastric fluid and the other pancreatic juice (mixed with duodenal secretions and bile). After base-line observations are made, the pancreas is stimulated by injecting secretin intravenously, and postinjection samples are obtained. The total volume and the bicarbonate and enzyme content of the specimens taken before and after secretin injection are then determined and compared with known normal values.

Other tests of pancreatic function include studies of the enzyme and fat content of the stool and determination of starch tolerance. The latter test is designed to measure the activity of the pancreatic carbohydrate-splitting enzymes as indicated by changes in the blood sugar after ingestion of a standard starch meal.*

X-ray studies are often helpful in both acute and chronic pancreatic diseases. In acute pancreatitis, one may find evidence of associated biliary tract disease, as indicated by radiopaque gallstones. Sometimes there is displacement of the stomach or colon by a pancreatic mass. In the chronic conditions, evidence of biliary tract disease and areas of calcification within the pancreas are common. Occasionally the duodenum is displaced or the duodenal loop is widened by a pancreatic mass. X-ray examination of the pancreatic duct system cannot be made except during operation.

*The level of radioactivity of the blood and stool after ingestion of a standard amount of I^{131} labeled fat is also an index of pancreatic enzyme secretion. The test is still in the experimental stage of its development but may soon be used clinically.

indicate that a lesion of the head of the pancreas is the cause. Benign diseases of the biliary tract often closely simulate pancreatic cancer.

6. Emotional disturbances are associated with abdominal pain. Patients with pancreatic cancer, and occasionally those with chronic pancreatitis, often complain of depression, crying spells, insomnia and inability to concentrate—symptoms which may be ascribed by the

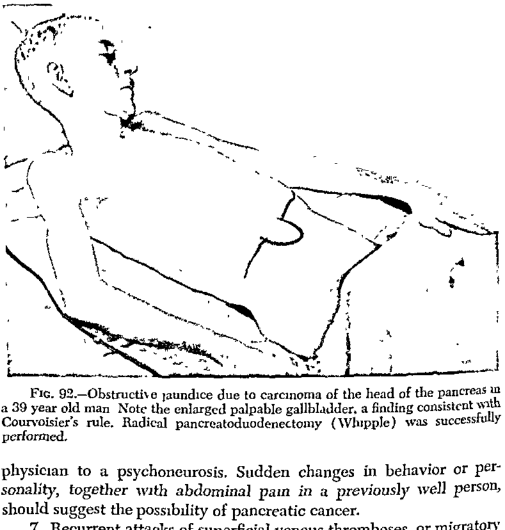

FIG. 92.—Obstructive jaundice due to carcinoma of the head of the pancreas in a 39 year old man. Note the enlarged palpable gallbladder, a finding consistent with Courvoisier's rule. Radical pancreatoduodenectomy (Whipple) was successfully performed.

physician to a psychoneurosis. Sudden changes in behavior or personality, *together with abdominal pain in a previously well person*, should suggest the possibility of pancreatic cancer.

7. Recurrent attacks of superficial venous thromboses, or migratory thromboses, are often associated with carcinoma of the pancreas and occasionally with carcinoma of the alimentary tract or the lung. A hypercoagulability of the blood, ascribed to trypsin in the circulation, is believed to cause spontaneous venous clotting.

There are no important physical signs in early chronic pancreatic

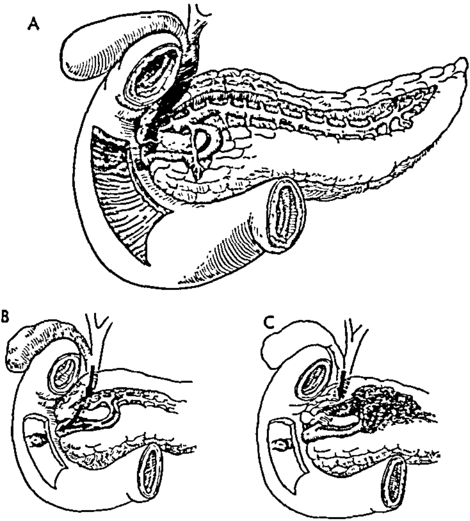

FIG. 93.—The pancreas and bile ducts in pancreatitis. *A*, the pancreas and its duct system. Note the papilla of Vater on the posterior medial wall of the second portion of the duodenum. *B*, the "common channel" between the common bile duct and the main pancreatic duct. Reflux of bile and activation of pancreatic ferments are possible when closure of the terminal common duct occurs from edema, stone or spasm of the sphincter of Oddi. This is the basis for the "common channel" theory of pathogenesis of acute pancreatitis. *C*, pancreatic edema, hemorrhage or necrosis follow activation of proteolytic and lipolytic enzymes within the pancreas by interaction with bile.

DISEASES OF THE PANCREAS

In this discussion, only conditions of surgical importance will be considered. These include: acute and chronic pancreatitis, cysts of the pancreas, cancer of the pancreas and islet cell tumors.

Acute Pancreatitis

The pancreas may become involved in an acute inflammatory process in which localized or diffuse edema, hemorrhage or necrosis is the predominant pathologic finding. The type of reaction which appears probably depends more on the severity of the specific insult than on the mechanism of its production. In other words, acute edematous pancreatitis, acute hemorrhagic pancreatitis and acute gangrenous pancreatitis are most likely not different diseases but variations of the same pathologic process.

In *acute pancreatic edema* a segment, or in some instances the entire gland, is swollen. Small areas of subperitoneal hemorrhage and fat necrosis often develop. The clinical manifestations tend to be less severe than in other forms of pancreatitis, and usually the patient recovers but often suffers repeated attacks. In *hemorrhagic and gangrenous forms of acute pancreatitis*, the changes are more extensive and usually life threatening. There is widespread digestion of vessels and bleeding into and around the pancreas. The entire gland may undergo necrosis and liquefaction. Areas of fat necrosis develop in both the retroperitoneal and intraperitoneal regions; and an abundance of brothlike, enzyme-rich fluid collects in the abdominal cavity. The disease may be reversible or rapidly fatal. It is always serious, and the prognosis must be guarded.

The pathogenesis of acute pancreatitis is incompletely understood, but intrapancreatic activation of digestive enzymes and autodigestion of the pancreas appears to be basic. It is easy to understand how, once initiated, the process can become self-perpetuating until the entire gland (and the individual) is destroyed. How the process is set off, however, is another matter. It is well known that injuries to the pancreas (contusion, operative trauma, etc.) can precipitate acute pancreatitis. Likewise, infections such as mumps and acute hepatitis may be complicated by acute pancreatitis, but infections are rarely the primary cause of acute pancreatitis. Spontaneous pancreatic hemorrhage or embolic occlusion of the pancreatic arteries may also

to the islands of Langerhans; and the serum bilirubin is above normal if drainage of the common duct is impaired.

The serum amylase determination is, by all odds, the most important diagnostic measure. It should be secured immediately in all instances of acute abdominal pain in which the possibility of acute pancreatitis exists. Levels above 500 units (Somogyi method) are to be expected, and 1,500 units or more are not uncommon. By the third or fourth day the values are usually normal (80–150 units). The urinary diastase and serum lipase tests may be of confirmatory diagnostic value.

Differential Diagnosis.—Conditions which must be differentiated from acute pancreatitis include: perforated peptic ulcer, acute intestinal obstruction, acute obstructive cholecystitis and acute appendicitis. Mesenteric vascular occlusion or strangulation of the bowel due to volvulus may closely mimic the clinical picture of acute pancreatitis. Acute diseases of the biliary tract, especially impaction of a common duct stone, can duplicate the picture of acute pancreatitis and may in fact be associated with acute pancreatitis on the basis of bile reflux through a common channel. Other catastrophic illnesses which simulate acute pancreatitis are: dissecting aortic aneurysm and coronary infarction.

Treatment.—The treatment of acute pancreatitis is nonoperative except (1) when the diagnosis is in doubt, (2) when there is an associated condition which aggravates the pancreatitis (e.g., common duct stone) or (3) when complications of pancreatitis, such as abscess, cyst or intestinal obstruction, develop.

The nonoperative treatment of acute pancreatitis includes:

1. Prevention and treatment of shock. Blood transfusions are necessary.
2. Correction of fluid and electrolyte imbalance by parenteral means.
3. Control of ileus and prevention of distention by nasogastric suction.
4. Relief of pain by use of Demerol® rather than morphine. The latter causes spasm of the sphincter of Oddi. Splanchnic nerve block with procaine often provides effective pain relief and appears to hasten recovery through release of local vasospasm.
5. Depression of pancreatic function through avoidance of oral feedings, removal and neutralization of gastric acid and administration of anticholinergic drugs.

precipitate acute pancreatitis. It has also been shown that obstruction to the duct system (edema, stone or tumor) while the gland is actively secreting will produce acute pancreatitis. Recently, acute pancreatitis has been produced experimentally by the induction of a local Schwartzmann reaction.

The "common-channel" theory (Opie, 1901) is applicable to many, but not all, instances of acute pancreatitis (Fig. 93). In about 60 per cent of all persons the terminal portions of the common bile duct and the main pancreatic duct unite before they enter the duodenum. Any obstruction to the common outlet—whether it be due to a gallstone, local edema or spasm of the sphincter of Oddi—will in fact convert the duct systems into a continuous tube or a common channel. Under these circumstances, bile can flow into the pancreatic ducts, activate enzymes and thus initiate autodigestion. In view of the fact that, percentage-wise, the arrangement of the duct systems favors reflux of bile, it is surprising that pancreatitis does not occur more often than it does.

Clinical Findings.—Acute pancreatitis occurs in both sexes and at any age but is most common in females during middle life. Often there is a history of biliary tract disease, alcoholism or previous attacks of pancreatitis.

The onset of the disease is usually abrupt and may follow a heavy meal. There is sudden upper abdominal pain, often excruciating in degree, which is referred to the back or scapular areas. Nausea, vomiting and retching are common. In severe pancreatitis the signs of shock soon appear. Initially, the abdomen is flat, and tenderness and muscle spasm are localized to the middle or upper zones. The bowel sounds decrease or disappear, and soon distention develops from paralytic ileus. Abdominal tenderness and muscle guarding may become diffuse. Sometimes a mass can be felt in the pancreatic area, and on rare occasions a bluish discoloration is observed about the umbilicus (Cullen's sign) from retroperitoneal extravasation of blood. A mild degree of jaundice frequently develops as a result of regional swelling around the common duct

The laboratory studies often confirm the diagnosis. There is moderate to marked leukocytosis; the serum amylase is increased for a short time (twenty-four to forty-eight hours) and then falls rapidly; the serum calcium is lowered (tetany may appear), owing to the loss of ionizable calcium into areas of fat necrosis, the blood sugar is elevated and the urine contains sugar if there is damage

diabetes and control frequent stools; but usually pain persists and narcotic addiction becomes a problem. If associated disease of the biliary tract exists, it should be treated surgically, with the hope that if there is a common channel condition it can be corrected. In some instances, division of the sphincter of Oddi (sphincterotomy) may prove effective. Occasionally, resection of a portion, or rarely the entire pancreas, is necessary. Several other operations have also been proposed for chronic pancreatitis, but none have proved very satisfactory.

Cysts of the Pancreas

Both true and false (or pseudo) cysts develop in the pancreas. True cysts are those lined with epithelium; they may be the result of obstruction to the duct system with continued secretory activity proximal to the obstruction, or they may be produced by a multiloculated tumor such as a cystadenoma. Pseudo cysts have a fibrous or granulation-tissue lining and result from traumatic or spontaneous pancreatic hemorrhage and necrosis. They are filled with fluid containing pancreatic secretion, blood, inflammatory exudate and necrotic tissue. The cyst fluid often contains enzymes in high concentration; and if the cyst communicates with a major duct, external drainage will be followed by a prolonged discharge of pancreatic juice.

True cysts of the pancreas cause symptoms only as they expand and compress regional structures. Pseudo cysts associated with acute or chronic pancreatitis cause pain, nausea, vomiting and a slowly developing abdominal mass which may reach the size of a football. Obstructive jaundice and diabetes often complicate the picture.

The clinical diagnosis is established by x-ray studies, which serve to locate the mass in the pancreatic area and to exclude tumors of stomach, colon, liver, kidney and adrenal origin. Final diagnosis usually depends on abdominal exploration.

Surgical treatment of pancreatic cysts includes: excision, pancreatic resection, external drainage (marsupialization) and internal drainage (cystogastrostomy or cystoenterostomy).

Carcinoma of the Pancreas

Cancer of the pancreas is a relatively common type of visceral neoplasm. It usually occurs beyond the age of forty and is three times

6. Prevention and control of secondary infection of the damaged tissues by use of broad-spectrum antibiotics.
7. Prevention and treatment of hypocalcemic tetany.
8. Treatment of diabetes, should it exist.

When acute pancreatitis is unexpectedly encountered at operation, the pancreas should not be disturbed unless localized areas of necrosis requiring drainage are found. The biliary tract should be examined for associated disease and, if necessary, drainage of the gallbladder or common duct instituted. More extensive operations in already critically ill patients often lead to death. The nonoperative measures outlined above should be continued during the postoperative period.

Chronic Pancreatitis

Recurrent attacks of mild or moderate acute pancreatitis appear to cause fibrosis, constriction of ducts, destruction of the glandular tissue and formation of cysts and areas of calcification in the pancreas. The entire gland eventually becomes hard, nodular and enlarged. The secretory function and often the hormonal function become impaired. Occasionally, narrowing of the common bile duct, in its course through the pancreas, results in obstructive jaundice. The clinical course in chronic pancreatitis is characteristically protracted, progressive and more or less disabling.

The patient complains of chronic and recurrent abdominal pains, usually aggravated by eating and relieved by vomiting. There is persistent abdominal soreness and tenderness. The deficiency of pancreatic secretion results in inadequate digestion and absorption of food, with weight loss and failure to gain. Diabetes is sometimes discovered before the diffuse nature of the pancreatic disease is recognized. The clinical findings may also indicate the presence of associated biliary tract disease; and at operation extensive changes in the pancreas, as well as in the bile ducts, will often be encountered.

The diagnosis of chronic pancreatitis is established by the history; the presence, sometimes, of an abdominal mass (enlarged pancreas or pancreatic cyst); the stool changes; the secretin test for pancreatic function; and the x-ray findings. The latter may reveal pancreatic calcification, deformities of the duodenum or cysts displacing the colon or stomach.

The treatment of chronic pancreatitis is most difficult. Medical measures may serve to improve the patient's nutrition, control the

depend on exclusion and suspicion. This is the time to consider operation. In many cases the main diagnostic problem concerns the differentiation of malignant from benign *obstructions of the common duct*. Sometimes the diagnosis can be correctly made on the basis of the existing clinical and laboratory picture; but because this, too, is subject to serious errors, exploratory operation *is indicated in most instances*. (See Chapter 18, on The Liver.)

The surgical treatment of cancer of the pancreas is, for the most part, palliative. *Occasionally, patients with cancer of the pancreatic* head have been cured by radical resection of the proximal pancreas and adjacent structures (duodenum, distal common bile duct and distal stomach) and *reanastomosis, or the Whipple operation. The morbid*ity and mortality which attend such an operation are understandably high; and, furthermore, operation is frequently not applicable. The *disease is often incurable by the time operation is undertaken, or the* patient is unfit for such an extensive operation. Cancer of the body and tail are somewhat more amenable to surgical resection; but here, *again, often the disease has already spread beyond the pancreas.*

Palliative surgical treatment consists in providing a by-pass for the obstructed bile passages. This can be accomplished by *cholecystogastrostomy, cholecystoduodenostomy or cholecystoenterostomy.* The patient is thereby relieved of jaundice and the intractable itching which often accompanies it. Although some improvement in his general condition usually results from operation, *pain may continue* and survival time is relatively short, averaging about six to eight months.

Islet Cell Tumors

Islet cell tumors are benign, or rarely malignant, adenomas of the islands of Langerhans. They are single or multiple, small (1–2 cm.) circumscribed, highly vascularized masses, most commonly located in the tail or body of the pancreas. They occur at all ages, but most often in young or middle-aged people. Islet cell tumors may be functional, producing insulin in excess of physiologic needs, or they may be nonfunctional.

Often the history is quite characteristic of hypoglycemic shock. The patient may complain of recurrent attacks of weakness, confusion, fainting or epileptiform convulsions, which develop during the fasting *state and are relieved by eating.* For any one patient the attacks tend

more frequent in men than women. The tumor develops in the pancreatic head in about 75 per cent of cases and in the body and tail in 25 per cent. Initially there is an area of localized woody hardness; but as the growth expands and infiltrates, it causes obstruction to the duct system, destruction of the glandular tissue and, if strategically located with respect to the common bile duct, obstructive jaundice. The disease spreads by contiguity, as well as through lymphatic and vascular channels to the peripancreatic tissues, the regional and distant nodes and the liver.

Early in the course of pancreatic cancer there may be little to suggest the correct diagnosis. Pain is highly significant, but it occurs in only about half the cases. The pain is often similar to that of chronic pancreatitis; and, in fact, the clinical aspects of both diseases are frequently so much alike that the diseases can be distinguished only by means of biopsy or necropsy. The pain is dull, boring and deep seated. It often goes "straight through to the back" or is referred to the scapular areas. The nocturnal character of the pain and the positions of relief have already been mentioned. Indigestion, nausea, vomiting, loose stools, rapid and severe weight loss are usual. Migratory phlebitis, diabetes and psychic aberrations should suggest the possibility of pancreatic cancer.

Often the diagnosis is made after months have passed and when jaundice appears (tumors of the pancreatic head) or when the signs of malignant disease become unequivocal. In some instances the diagnostic mystery could have been solved much earlier, and the patient could have been spared much mental and physical anguish, by a more thoughtful investigation of his complaints and by early recourse to abdominal exploration

There is little in the way of objective findings in early pancreatic cancer. The abdominal mass is rarely palpable. There may be slight icterus and obvious malnutrition. The x-ray studies may be helpful, but normal findings do not exclude this disease. Distortion of the duodenal papilla is a clue to carcinoma of the ampulla of Vater. Blood in the stool from ulceration into the duodenum, associated with obstructive jaundice, points strongly to ampullary (pancreatic) carcinoma.

The secretin test often is an aid to diagnosis if properly made. Determinations of the fat and enzyme content of the stool and the starch tolerance are indicated in obscure diagnostic problems.

When all the evidence has been gathered, the diagnosis may still

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to follow the same pattern and to develop under similar circumstances. That is, an attack may always occur in the early morning hours, after a missed meal or after strenuous exercise. Commonly the disorder is erroneously called "idiopathic" epilepsy, after which the investigation is terminated and anticonvulsive drugs are given indefinitely. Such management would appear to represent a serious breach of medical practice, because hyperinsulinism due to islet cell tumor, in contradistinction to most other pancreatic conditions, is curable.

The diagnostic triad of Whipple is worth remembering: (1) central nervous system symptoms, which may be neuromuscular, vasomotor or psychic, often resembling epilepsy; (2) onset of symptoms during the fasting state, associated with blood sugar levels below 50 mg./100 ml.; and (3) immediate recovery following oral or parenteral administration of sugar.

Hyperinsulinism must be differentiated from hypoglycemia due to other causes, such as hepatic disease, endocrine disturbances (pituitary, adrenal, thyroid) and functional hypoglycemia. Often the diagnosis must be made by exclusion and proved by exploration.

Medical treatment may be adequate in mild cases; but with progression of symptoms, operation becomes necessary. Exploration of the pancreas in search of one or more islet cell tumors is indicated in all but the mildest cases. The tumor or tumors are often visible on the surface of the pancreas or can be felt within the substance of the gland. Infrequently, diffuse hyperplasia of the islets, rather than isolated tumors, is encountered. Partial resection of the pancreas is indicated under these circumstances, but the results of such treatment are not so good as those which follow removal of a functioning tumor.

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Accessory spleens are ectopic aggregations of splenic tissue commonly found in the region of the splenic hilus, less often in other portions of the embryonic dorsal mesogastrium in which the spleen is formed. Accessory spleens are similar in all respects to the spleen except for size and shape. They are single or multiple, small, round, reddish masses which vary in size from a few millimeters to several centimeters. The importance of accessory spleens lies in the fact that they are functional and must be removed when splenectomy is undertaken for hematologic disorders.

The spleen is covered by a thin capsule of peritoneum and fibrous tissue which is continuous with the splenic stroma. The fibrous trabeculae which divide the spleen into compartments contain the large vessels which enter the parenchyma. The trabeculae also contain smooth-muscle cells which give the spleen a certain degree of contractibility.

The splenic parenchyma consists of a white pulp (malpighian corpuscles), which surrounds the terminations of the splenic arteries, and of a red pulp, which communicates with splenic veins. Lying between the arteries and veins are arterioles, capillaries, venous sinuses and venules. The spaces between the venous sinuses of the red pulp are filled by the splenic cords containing reticuloendothelial cells, lymphocytes, monocytes, macrophages and leukocytes. The relationship between the circulating blood elements and the specialized fixed and wandering cells is an intimate one, and here much of the work of the spleen is performed.

There has been much discussion as to whether the splenic circulation is closed or open, or both closed and open. The closed-theory proponents maintain that the blood flows directly from the arterial capillaries to the splenic sinuses and to the veins. The open-theory proponents maintain that the sinuses are fenestrated and that blood passes back and forth through the fenestrations into the pulp cords. The proponents of the combined open and closed theory maintain that the spleen has a divided circulation which permits red cells and plasma to pursue different pathways. Red cells can be stored in the sinuses, and plasma can exude freely into the pulp cords. Thus, sequestration of red cells, filtration of plasma and diapedesis of cells can occur. The evidence appears to favor a combined type of splenic circulation, but this issue has not yet been settled.

The functions of the spleen are:

Blood Production.—The spleen is an important blood-forming organ during embryonic life, but later it forms only lymphocytes. In

CHAPTER 20

The Spleen

THE SPLEEN is a soft, bluish red, fist-sized organ located high and deep in the left side of the abdominal cavity. It lies in apposition to the posteroinferior surface of the diaphragm, the costal arch and the stomach. Its upper, smooth convex surface is free, while its lower, concave hilar surface is fixed by peritoneal ligaments. The hilus lies in close relationship with the tail of the pancreas. The main splenic vessels pass from the superior surface of the pancreas directly into the splenorenal ligament and thence to the hilus. The lower border of the spleen lies near the retroperitoneally located left adrenal and kidney.

The weight of the spleen varies according to the amount of blood that it contains, but the weight averages about 150 Gm. In consistency, the spleen is similar to that of other soft viscera, resembling most closely the liver. Although anchored by peritoneal attachments, the spleen is quite mobile and subject to shifting and displacement from internally or externally applied forces; and therefore, several factors make the spleen particularly vulnerable to injury. These include: its superficial location and its consistency, friability and mobility.

Three double-layered peritoneal folds constitute the main splenic ligaments and are attached at the hilus to form the splenic pedicle. From the front the ligaments are: (1) the presplenic fold, an avascular sheet lying between the gastrocolic ligament and the hilus; (2) the gastrosplenic ligament, containing the short gastric vessels, which runs between the greater curvature of the stomach and the hilus; and (3) the splenorenal ligament, containing the splenic artery and vein, which is attached to the tail of the pancreas and the hilus. Several smaller and less important peritoneal folds are attached to the diaphragm, colon and spleen. All of these structures must be divided and their vessels ligated in splenectomy.

of splenic enlargement in the latter disease is attributed to the fact that the platelets are so small and so easily destroyed that they cause little reaction in the spleen.

The term "hypersplenism" has been applied to those conditions in which the spleen, by virtue of a functional increase of some normal processes, produces hematologic disturbances. Hypersplenism can be either of primary or of secondary origin. Primary hypersplenic conditions tend to develop in the absence of demonstrable splenic, bone

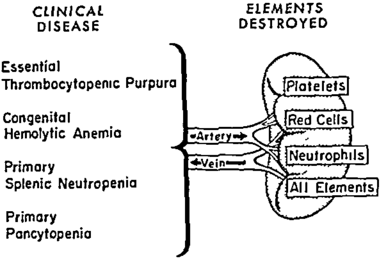

FIG. 94.—Role of spleen in "primary" hypersplenic syndromes. (From Zollinger, R. M., *et al.*: Surgical aspects of hypersplenism, J.A.M.A. 149:24, 1952.)

marrow or systemic disease. The secondary hypersplenic syndromes are hematologic disturbances resulting from splenic disease and splenomegaly. Secondary hypersplenism is most commonly associated with cirrhosis of the liver, Hodgkin's disease, the leukemias, sarcoidosis and multiple myeloma. Splenectomy may also influence the blood picture favorably in secondary hypersplenism, but the results are often less pronounced.

When, as a result of bone marrow aplasia, the spleen reverts to its embryonic function of producing myeloid elements (agnogenic myeloid metaplasia), it often constitutes the chief source of the blood elements. Removal of the spleen under these conditions may lead to failure of hemopoiesis, and death. Agnogenic myeloid metaplasia is therefore an important contraindication to splenectomy.

Congestive splenomegaly with secondary hypersplenism is believed to be the result of portal hypertension. Portal hypertension is

certain disease states (e.g., myeloid leukemia), the spleen reverts to its original function of complete hemopoiesis and produces red cells, white cells and megakaryocytes.

Regulatory Influence on the Blood Elements.—The spleen normally is the site of destruction of worn-out blood cells, and it also exerts a restraining influence on the bone marrow, possibly through specific humoral substances. Removal of the spleen is followed by temporary leukocytosis, thrombocytosis and, occasionally, erythrocytosis.

Reticuloendothelial and Lymphoid Functions.—These functions include: phagocytosis, bacterial and particle fixation, antibody production, hemoglobin degradation and iron storage.

Blood Reservoir.—Storage of blood and mobilization of stored blood in response to adrenergic stimuli occur in animals but do not appear to be important in man.

CLINICAL CONSIDERATIONS

Removal of the spleen in an otherwise healthy person results in no permanent impairment of health. The blood levels gradually return to normal, and there are no observable changes in the patient's resistance to infection or in his response to trauma and shock. The remaining reticuloendothelial and lymphoid tissues are apparently capable of taking over the functions of the spleen.

Removal of the spleen for certain hematologic diseases frequently causes the blood findings to revert to normal. This is often strikingly exemplified by the response which follows splenectomy for the primary hematologic conditions in which no local (splenic or bone marrow) or systemic disease is recognized (Fig. 94). Thus, removal of the spleen for platelet deficiency (idiopathic thrombocytopenic purpura), red cell deficiency (congenital hemolytic anemia), neutrophil deficiency (primary neutropenia) or deficiency of all the myeloid cells (primary pancytopenia) often results in the cure of the hematologic disease. In these conditions it is believed that, for some reason, the spleen has become hyperactive and either sequestrates and destroys excessive numbers of blood elements or limits the maturation and discharge of blood elements from the bone marrow.

The hyperactive or hyperfunctioning spleen, associated with the foregoing clinical conditions, is usually enlarged and palpable (splenomegaly). An important exception is noted in idiopathic thrombocytopenic purpura, in which splenomegaly is uncommon. The absence

red cell fragility, bleeding and clotting times, clot retraction, prothrombin level and the Coombs test. The latter is an aid in differentiating acquired from congenital hemolytic anemia.

Liver function studies are indicated when the clinical picture suggests combined hepatic and splenic disease, as, for example, in congestive splenomegaly with cirrhosis. Under these circumstances the esophagus should also be examined for varices by radiographic and endoscopic technics. The status of the portal circulation can be investigated in selected cases of splenomegaly by measuring portal venous pressure and by demonstrating the portal system radiographically (splenoportography). This is accomplished during operation or occasionally by introducing a needle directly into the spleen percutaneously. These are special and potentially dangerous investigative technics and should only be performed by those who are experienced in their application.

CLINICAL CONDITIONS

The most common surgical conditions of the spleen will be considered. These include: traumatic rupture of the spleen, the hypersplenic syndromes and congestive splenomegaly.

Traumatic Rupture of the Spleen

Although the spleen is located in a relatively well-protected portion of the abdomen, it may be lacerated or ruptured by apparently slight trauma. Most often, splenic ruptures are the result of direct blows to the margin of the left rib cage, which overlies the spleen. Commonly there will be local signs of contusion or fractured ribs. Sometimes injury to the spleen results from indirect trauma, as, for example, from a fall, or a blow from a steering wheel. Not infrequently, the spleen may be lacerated by a penetrating object, such as a bullet, knife or even the jagged end of a fractured rib.

The injury to the spleen may cause tearing of its capsule, laceration of its vascular pedicle or disruption of the splenic pulp. With laceration of the capsule or pedicle, bleeding occurs into the peritoneal cavity. If bleeding is of severe proportions, the resulting clinical picture is that of a catastrophic abdominal condition. If the bleeding is less severe but persistent, the onset of signs of hemorrhage may be delayed but the symptoms may be expected to be persistent and pro-

caused by sustained blockage of the portal blood flow. The portal block may be caused by scarring, thrombosis, inflammation or compression proximal to the liver or within the liver. In congestive splenomegaly, one or more blood elements are depressed, the spleen is enlarged, and abnormal venous collateral channels pass between the portal and systemic venous systems.

Enlargement of the spleen is also caused by a large number of inflammatory, neoplastic and infiltrative conditions. Abscesses of the spleen are uncommon, or at least are uncommonly recognized. Tuberculosis is the usual cause of splenic abscesses. Splenic enlargement of varying degree is, however, encountered in a number of systemic infections, including chronic malaria, typhoid fever, infectious mononucleosis, Felty's syndrome and sarcoidosis.

Splenomegaly due to tumors of the spleen is also uncommon. Both primary and metastatic neoplasms of the spleen occur, but their rarity suggests that the spleen possesses a special type of immunity. Cysts of the spleen develop from parasitic infestation (echinococcus) or from organization of splenic hematomas or abscesses. The diagnosis may be apparent if calcium is found in the cyst wall on x-ray examination.

There are a number of metabolic disturbances which are associated with splenomegaly. These conditions include: Gaucher's disease, Niemann-Pick's disease, Hand-Schüller-Christian's disease and Letterer-Siwe's disease. The splenomegaly is often extreme and results from the storage of excessive amounts of lipids or polysaccharides in the reticuloendothelial cells. There is associated involvement of other elements of the reticuloendothelial system, including the liver, lymph nodes and bone marrow, as well as hematologic changes of secondary hypersplenism and pronounced clinical changes. Splenectomy may be of palliative value but is infrequently indicated in these diseases.

The presence of disease of the spleen may be suggested by a history of splenomegaly, by abnormal bleeding into the skin or from the body orifices, by chronic anemia with recurrent hemolytic crises and jaundice or by unusual sensitivity to infections associated with leukopenia or pancytopenia. In most conditions, there will be enlargement of the spleen. The margin of the spleen may be palpable at the costal margin or may, in some instances, extend into the pelvis.

The laboratory studies are of great importance to diagnosis. In complicated cases the help of a hematologist should be enlisted. Hematologic studies often include: peripheral blood and bone marrow smears, thrombocyte counts, search for spherocytes, determination of

crease in the blood pressure. The respiratory rate is often elevated as a result of impaired ventilation from pain, fractured ribs, etc. With continued bleeding and prior to the onset of shock, the temperature tends to rise slightly.

The laboratory findings may or may not be helpful. Leukocytosis is to be expected after any injury and cannot be regarded as evidence for or against splenic rupture. The red cell count, hemoglobin and hematocrit values are normal until hemodilution occurs a few hours after blood loss; then they tend to fall. Serial determinations should be made at two, three or four hour intervals when the patient is under observation for suspected visceral injury.

X-ray studies are often helpful. If fractured ribs are found over the splenic region, the suspicion of splenic rupture is reinforced. Occasionally, an associated hemothorax, pneumothorax or rupture of the diaphragm may be indicated by the chest films. Sometimes the normal splenic shadow is larger than it should be, and the air-containing stomach and/or colon are found to be displaced. Infrequently, air in the peritoneal cavity due to perforation of a hollow abdominal organ will be associated with splenic rupture.

Aspiration of the peritoneal cavity for free blood is sometimes advised when the diagnosis of rupture is in doubt. This diagnostic procedure is only helpful when blood is found on aspiration, and it is, of course, potentially harmful.

Splenectomy is the only treatment for splenic rupture. The operation should, if possible, be performed before the patient has passed into the stage of severe shock. Because the diagnosis cannot always be established with certainty, often abdominal exploration is in order when the signs suggest visceral injury with intraperitoneal hemorrhage but the nature and extent of the injury are unknown.

Matched blood must be secured and administered in amounts sufficient to restore and maintain circulatory dynamics. A nasogastric tube should be introduced and suction applied to determine the presence of blood, to empty the stomach and to avoid the danger of aspiration of vomitus under anesthesia. Ideally, the patient should be out of shock at the time of operation; but when there is massive hemorrhage, correction of shock depends on the control of bleeding. In these circumstances, therefore, emergency splenectomy constitutes an all-important resuscitative measure.

The operation is performed under general anesthesia. The splenic pedicle is isolated, its vessels ligated, and the spleen removed. Free

gressive as the left upper quadrant hematoma enlarges. When bleeding occurs into the spleen and within its capsule, either the bleeding may stop spontaneously or catastrophic hemorrhage may follow delayed rupture, days or weeks after injury.

Splenomegaly from any cause increases the danger of splenic rupture. Rupture may follow minor abdominal injury and sometimes occurs spontaneously. Patients with splenic enlargement should be warned about this hazard.

The signs and symptoms of rupture of the spleen depend on the degree of fragmentation, the severity of the local and diffuse peritoneal irritation due to blood and the amount and rate of blood loss. Obviously, the clinical picture is subject to great variation, but the possibility of splenic rupture must be considered in all patients who suffer abdominal, thoracic or combined abdominothoracic injuries.

There will usually be a history of direct or indirect injury to the splenic area. Following the injury, which is not necessarily severe, the patient appears to recover, then passes into a period of "delusive calm" and later exhibits the signs of progressive hemorrhage and shock.

Initially, there is pain in the left upper quadrant and lower chest, aggravated by breathing, coughing or turning. The pain is often referred to the left shoulder (Kehr's sign) over the phrenic nerve from the left subdiaphragmatic area. Nausea and vomiting are not likely to be prominent symptoms except when associated injuries to other abdominal organs exist. There is tenderness and splinting of the left side of the chest if direct injury to the splenic region has occurred. Fractured ribs may be palpable. Superficial abrasions, contusions and hematomas of this region are commonly observed.

The abdominal findings may not be too unusual soon after the injury has been sustained. Tenderness and muscle spasm in the upper abdomen, particularly on the left, is often localized, then later becomes diffuse. The abdominal muscles, however, are not rigid, as they are in perforated peptic ulcer, because blood is much less irritating to the peritoneum than gastric secretions. Some degree of rebound tenderness will be present. If a hematoma forms and enlarges, a mass may be felt in the left upper quadrant. Usually the bowel sounds become hypoactive and disappear as blood spreads throughout the peritoneal cavity.

Except in massive hemorrhage, the onset of signs of shock are delayed, but those of impending shock may be recognized. They may consist of only a persistent increase in the pulse rate and a slight de-

it follows an acute infectious disease. Spontaneous cerebral and retinal hemorrhage are particularly serious complications. The spleen is not enlarged.

The diagnostic blood findings are:

1. Reduction in the level of blood platelets
2. Prolonged bleeding time
3. Prolonged clot retraction time

The bone marrow is normal or shows increased megakaryocytes and erythroid elements. Capillary resistance is decreased, as demonstrated by the appearance of petechiae with the Rumpel-Leede tourniquet test. The secondary purpuras must be carefully excluded if surgical treatment is to be of value.

The condition of the patient, rather than the acuteness or chronicity of the disease, must be the determining factor in planning treatment. Cortisone or ACTH may induce a remission and, in some instances, possibly a cure. Splenectomy (including all accessory splenic tissue) is the preferred treatment and usually results in prompt cessation of active bleeding, a decrease in the bleeding time, an immediate and sustained rise in the platelet count and disappearance of petechial hemorrhages and ecchymoses.

While the concept of hypersplenism as the basic mechanism in the development of this disease has much to support it, splenic hormones influencing hemopoiesis in the bone marrow, faulty maturation of the megakaryocytes or auto-immune platelet destruction may also be important factors.

Idiopathic Neutropenia

In idiopathic, or primary, splenic neutropenia, the spleen is enlarged and filled with large numbers of neutrophils in various stages of disintegration. The bone marrow may be normal or may show an increase in the myeloid series. The clinical manifestations of this condition are: a tendency to increased infections, fatigue, weakness, mouth ulcers and leg ulcers. The diagnosis is established by blood studies, including the bone marrow. Splenectomy may constitute effective treatment.

Idiopathic Pancytopenia

In idiopathic pancytopenia (primary splenic hematopenia), all the blood elements of bone marrow origin are decreased. The spleen

and clotted blood is evacuated; and after a search is made for injury to other organs, the abdomen is closed.

Congenital Hemolytic Jaundice

Congenital hemolytic jaundice (spherocytosis, primary hemolytic jaundice) is a chronic, often mild, disease characterized by persistent and variable jaundice, splenomegaly and anemia. Hemolytic crisis may occur. Gallstones (bilirubin type) are common. The diagnosis is confirmed by laboratory findings:

1. Spherocytes in the peripheral blood
2. Increased fragility of erythrocytes in hypotonic salt solution (normal, 0.3–0.5%)
3. High reticulocyte count and presence of immature red cells in the blood

It is probable that a hereditary defect in red cell generation in the bone marrow results in the production of spherocytes. The spleen removes and destroys the abnormal cells while the normal red cells "die" after a life span of about 120 days. Destruction of large numbers of spherocytes and possibly impaired liver function and overactivity of the bone marrow leads to jaundice. The jaundice is often chronic; but acute hemolytic crises may produce intermittent anemia, jaundice, abdominal pain, weakness, dyspnea, palpitation, etc.

The results from splenectomy are excellent in typical cases. Operation is inadvisable during acute hemolytic crises and should be deferred unless the serious condition of the patient demands it. Blood transfusions are withheld, if possible, during crises because *there is a possibility of aggravating the hemolytic process.* Properly grouped and cross-matched sedimented red cells suspended in saline may be lifesaving in some instances. ACTH or cortisone often induce a remission of the acute process.

Idiopathic Thrombocytopenic Purpura

In idiopathic, or primary, thrombocytopenic purpura, the patient gives a history of widespread subcutaneous hemorrhages occurring spontaneously or after minor injury. Hemorrhage also occurs from the bowel, bladder, nose, uterus or other organs. The disease may appear suddenly in a severe form or may develop slowly and progress insidiously. It is more frequent in children than in adults, and sometimes

develop. This complication can be prevented or delayed by providing an artificial by-pass between the splenic vein and the left renal vein (splenorenal shunt) at the time of splenectomy.

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is enlarged, and the bone marrow is hyperplastic. A favorable response may follow the administration of ACTH or cortisone. Secondary hypersplenism due to a systemic disease must be considered and, if possible, ruled out before splenectomy is performed.

CONGESTIVE SPLENOMEGALY

Banti, in 1883, described a syndrome or disease (Banti's disease) characterized by anemia, splenomegaly, cirrhosis of the liver and ascites. Later he and other investigators believed that the syndrome was the end stage of splenic anemia. They considered that the process began with anemia and splenomegaly and then progressed to cirrhosis of the liver and finally to ascites. Recent studies suggest that this condition is the result of portal hypertension. Portal hypertension is produced by intrahepatic or extrahepatic blockage to portal flow, which may result from a variety of diseases. The most common causes of intrahepatic blockage are: portal cirrhosis and schistosomiasis; the most common causes of extrahepatic blockage and thrombosis, cavernous transformation and external compression of the portal vein (or splenic vein) by tumors or inflammatory processes.

Whatever the cause of portal hypertension, the spleen is affected and congestive splenomegaly with secondary hypersplenism results. The splenic sinusoids become dilated and engorged. There is an accompanying connective-tissue reaction which causes the spleen to become hard and its capsule to become thickened and fixed to the parietal structures by vascular adhesions. The pressure in the portal system (including the splenic vein) is elevated (two or more times normal of about 150 mm. of water), and numerous collateral channels between the portal and caval systems are demonstrable. The most important routes of collateral flow are to the veins of the abdominal wall (which form the caput medusa), to the esophageal plexus, to the pelvic veins and to the hemorrhoidal veins. The esophageal veins are of prime clinical importance because the varices which develop here often rupture and lead to massive hemorrhage and death.

Congestive splenomegaly cannot be treated effectively by simple splenectomy except in the rare instances where the obstruction is confined to the splenic vein. When the block is located in the portal vein or in the liver, as it is in most cases, splenectomy affords only a short period of clinical improvement, following which other complications of portal hypertension, including rupture of esophageal varices,

cal judgment or specialized treatment which he may be unable to provide is needed, he will request the help of those best able to give it.

Fortunately for both the patient and physician, nature's indomitable drive to survival and recovery (vis medicatrix naturae) includes many acute conditions of the abdomen. The patient will often recover without, with, or in spite of, treatment. In some conditions, unfortunately, the changes within the abdomen are irreversible, and without surgical treatment the patient will die—for example, the vicious cycle of intestinal obstruction, where operative treatment alone can reverse the process and save the patient's life. There are other abdominal conditions in which operation does no good (e.g., acute pancreatitis); and still others (extra-abdominal disturbances) in which operation is generally harmful. The decision, therefore, "to operate or not to operate" must be based on good clinical judgment, which comes with knowledge and experience.

Clinical Aspects

The entire clinical picture must be studied, and each bit of evidence weighed. Often there are bits of information, clues, perhaps hunches, which the clinician ties together to help him make a diagnosis which is most likely to be correct. Some of these clues pertain to the patient's age, sex, habitus, past and present history, appearance, emotional background, etc.

In the newborn, congenital anomalies often produce acute conditions of the abdomen. Disturbances of the gastrointestinal tract (usually obstruction) may result from imperforate anus, malrotation, volvulus, atresia of the gut, meconium ileus, tracheoesophageal fistula or diaphragmatic herniation. In the first few weeks of life, hypertrophic pyloric stenosis and megacolon may give rise to symptoms of obstruction. Later in infancy, ileocolic or ileoileal intussusception may occur. At this time, "colics" or infections (e.g., urinary tract) are common and may resemble acute surgical conditions. In childhood and young adult life the incidence of appendicitis is high. In adolescent and young adult females, hemorrhage from a ruptured graafian follicle may mimic acute appendicitis. In females, during the child-bearing period, ectopic pregnancy and infections of the genital tract are possible causes of lower abdominal pain. An ovarian cyst which has become twisted on its pedicle, pelvic inflammation, septic abortion and endometriosis are also considerations.

CHAPTER 21

Acute Surgical Conditions of the Abdomen

ACUTE CONDITIONS of the abdomen are generally produced by inflammatory, obstructive or vascular mechanisms and are manifested by abdominal pain, gastrointestinal symptoms and varying degrees of local and systemic reaction. They require urgent treatment, often including emergency operation. They range from easily recognized conditions to complex or obscure disturbances, the diagnosis of which may be most difficult. There are few specific tests or examinations which may be relied on to give clear-cut answers to these problems. Their urgency usually precludes prolonged investigations and deliberations and calls for prompt and decisive action. Although in some conditions x-ray studies are helpful, there are certain definite limitations to the use of radiographic methods. Similarly, most laboratory tests have limited diagnostic value.

The diagnosis of acute conditions of the abdomen, therefore, frequently resolves itself into arriving at a judgment based on the evidence derived from an accurate and complete history, a careful physical examination, a few laboratory tests and selected x-ray studies. From these data it is possible to analyze the clinical picture in terms of disturbed structure and function, to synthesize a working diagnosis or differential diagnosis and to prescribe rational treatment. While gathering the evidence, the physician must weigh the possibilities and probabilities in terms of specific disturbances. He must also consider the need for prophylactic, supportive and corrective measures, and he will time these measures so that investigation and treatment proceed concurrently. He must recognize the fallibility of his observations and interpretations and the variability of biologic phenomena. When criti-

Symptoms

Pain.—An analysis of the character of the pain is essential to diagnosis. The items which should be considered include: the mode of onset, location, intermittency or constancy, radiation, severity, accentuation and relief, and progression or regression. If analgesics (especially morphine) have been administered before examination, assessment is difficult. These drugs tend to cloud the patient's responses and to obscure the physical signs upon which the physician must base his diagnosis. Even in the absence of analgesics, there is

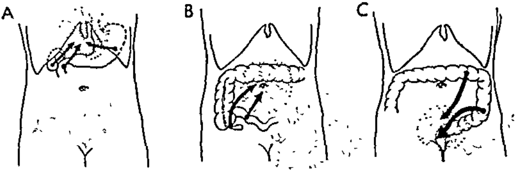

Fig. 95.—Primary zones to which visceral pain of alimentary tract origin is referred *A*, Zone 1 (epigastric area), from disturbances arising in the foregut derivatives. These include: stomach, duodenum (to the duodenal papilla), liver, bile ducts and pancreas. *B*, Zone 2 (umbilical area), from disturbances arising in the midgut [illegible] denal papilla, the [illegible] *C*, Zone 3 (hypo- [illegible] derivatives. These include: the left half of the transverse colon, the descending and sigmoid colon and the rectum.

great variation in pain patterns and individual responses, but some generalizations are possible.

As previously stated (in Chapter 13, on The Peritoneum), visceral pain is characteristically vague and poorly localized. It may be projected or referred into somatic zones through neural communications in the brain or cord. Pain of gastrointestinal origin, with few exceptions, is referred to three zones, located in the midline of the abdomen (Fig. 95). Disturbances of the stomach, duodenum, gallbladder and pancreas cause referred pain in the upper zone (epigastric); the small intestine, appendix and large intestine proximal to the midtransverse colon in the middle zone (umbilical); and the distal large intestine in the lower zone (hypogastric). On the other hand, when the parietal

In the adult male, perforated peptic ulcer and, in the adult female, acute obstructive cholecystitis are second only to acute appendicitis as causes of acute abdominal conditions. With advancing age (usually beyond forty), vascular and neoplastic diseases appear with increasing frequency and may precipitate acute abdominal pain. Embolic and thrombotic occlusion of the mesenteric vessels, although uncommon, should be borne in mind, particularly when the patient has evidence of cardiovascular disease.

Neoplastic diseases usually do not produce acute abdominal symptoms, but the physician is often misled unless he carefully evaluates the antecedent history. Carcinoma, especially of the large bowel, may precipitate acute colonic obstruction. Small-bowel obstruction is most often caused by strangulation in a hernia. If there is no obvious hernia, then a search should be made for an occult hernia in the femoral ring. If the patient has an abdominal scar and signs of intestinal obstruction, the odds will favor peritoneal bands or adhesions as the cause. A history of a previous intestinal obstruction due to adhesions strongly predisposes the patient to similar trouble.

The patient's build and temperament may be a clue to diagnosis. The patient with peptic ulcer is often an emotional, energetic, driving, thin male who has been subjected to unusual pressures. The patient with gallbladder disease commonly is an obese female who has had many children. But it should be remembered that acute gallbladder disease and perforated ulcer may occur in either sex and any body type. Patients with sigmoid diverticulitis are often fat and elderly. Acute pancreatitis occurs in both males and females. A history of pre-existing gallbladder disease or alcoholism should suggest this diagnosis

The past history may provide clues to direct diagnosis. Colicky pain is produced by smooth-muscle stretching or spasm and may arise from appendical, intestinal, biliary, tubal or ureteral obstruction. Periodic bouts of indigestion and pain, especially nocturnal, are common in peptic ulcer. Be alert in atypical appendicitis. Many patients with duodenal ulcer have been subjected to an unnecessary appendectomy. A history of "colitis" should suggest the possibility of cancer, diverticulitis or ulcerative or amebic colitis. Any disturbance in bowel function when accompanied by blood in the stool should suggest cancer first. All of the foregoing conditions may lead to acute abdominal symptoms when complications develop (perforation, obstruction, inflammation or hemorrhage).

peritoneum is irritated, pain, tenderness and muscle spasm appear and are localized to the area of somatic innervation. The abdominal signs in perforated peptic ulcer, for example, are usually generalized because diffusion of highly acid fluid causes intense irritation of the parietal peritoneal surfaces (somatic). In acute cholecystitis, visceral pain is usually referred to the upper zone of the abdomen and to the back or right scapula; but when parietal peritoneal irritation occurs (somatic), pain and tenderness are localized to the right upper quadrant. The pain of pancreatic inflammation is referred to the anterior upper zone and/or the back or interscapular area. Remember that the pancreas is a deep-lying retroperitoneal organ; and localized pain, tenderness and rigidity are not likely to be striking unless the anterior parietal peritoneum is also involved. The common pain patterns in acute abdominal conditions are shown in Figure 96.

The kidneys and ureters also lie retroperitoneally, and pain arising from diseases of these organs is usually referred into the flank, groin, genitalia or anterior aspect of the thigh. Abdominal pain and tenderness vary but are often not prominent.

Acute diseases of the female pelvic organs may cause lower abdominal pain, direct tenderness and pain on movement of the uterus and adnexa. There may be pain low in the back or radiating into the thighs. The signs and symptoms sometimes resemble those of appendicitis, diverticulitis, intestinal obstruction or urinary tract disease.

NAUSEA AND VOMITING—Slight nausea and vomiting may be of reflex origin and, as such, are nonspecific. Protracted vomiting is more significant and may result from physiologic or mechanical obstruction to the gastrointestinal tract; less commonly from metabolic imbalances, drug toxicity, infections and central nervous system disturbances. Vomiting may not be an outstanding feature of acute abdominal conditions caused by infection, hemorrhage or vascular occlusion unless complicated by intestinal obstruction, gastric dilatation, peritonitis or certain metabolic alterations.

CONSTIPATION OR DIARRHEA.—Constipation or diarrhea should be interpreted in terms of the entire clinical picture. With complete bowel obstruction, unrelenting constipation (obstipation) appears after the fecal matter beyond the block has been discharged. Commonly, obstruction is incomplete, and some gas and solid material will be expelled for a few days after the onset of symptoms of obstruction. This may lead to the conclusion that there is no obstruction,

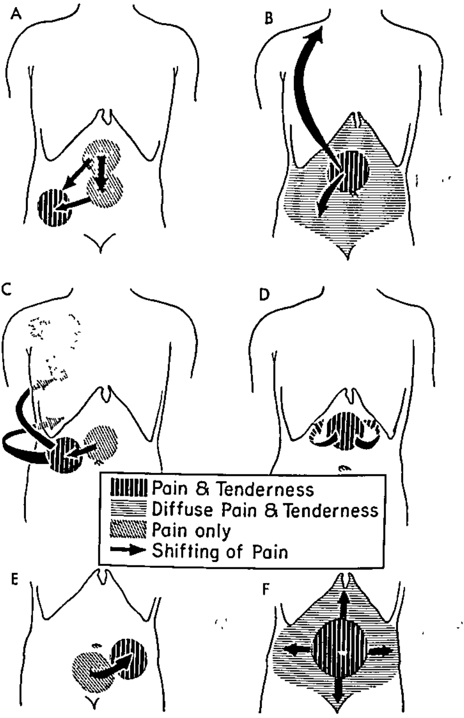

FIG. 96.—Common pain patterns in acute abdominal conditions *A*, acute appendicitis. *B*, perforated peptic ulcer. Note radiation of pain to the right shoulder. *C*, acute cholecystitis. *D*, acute pancreatitis. *E*, acute sigmoidal diverticulitis. *F*, acute obstruction of the blood supply of the bowel, as in mesenteric embolism, mesenteric thrombosis or strangulating intestinal obstruction.

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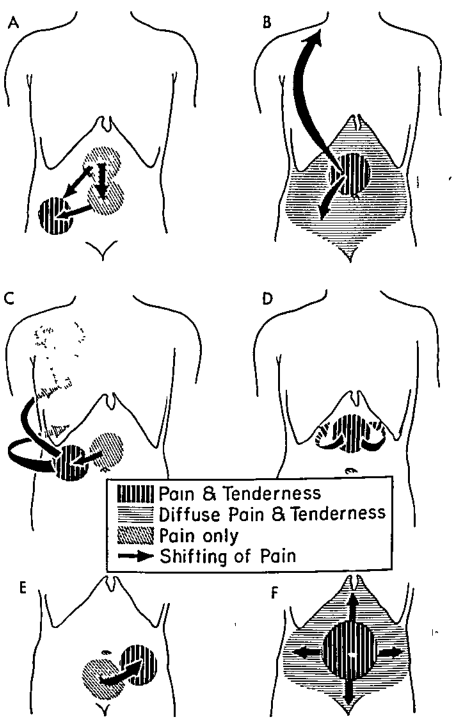

FIG. 96.—Common pain patterns in acute abdominal conditions A, acute ap-

thrombosis or strangulating intestinal obstruction.

temperature, pulse, respiration and blood pressure are recorded; this information provides a base line for later observations. Careful examination of the heart and lungs should be carried out, bearing in mind that disturbances in these organs may mimic acute abdominal diseases. At this time also, the functional capacity of these organs is assessed. If there is need for special laboratory tests, x-rays, electrocardiograms or consultations, the machinery for securing them should be set in motion.

The abdomen must be exposed completely for examination. The examiner's hands should be warm. A calm, reassuring and sympathetic approach on the part of the physician is helpful. He should ask the patient "Where is the pain?"and should tell him to "point to it with one finger." This may give important clues. The examiner should use special care when studying these areas.

Inspection of the abdomen often reveals significant surgical scars, hernias, striae, prominent veins, distention, bulging in the flanks, masses, discoloration or visible peristalsis. If inspection is cursory and the immediate attention is directed toward palpation, important signs may be overlooked. *Take time to look and think, as well as to feel and think.*

The next step is systematic palpation of the abdomen, beginning at a distance from the area of maximal tenderness and alternately testing and comparing each region with the opposite side. In the general abdominal survey, a search should be made for areas of tenderness, muscle spasm or induration, and then any specific areas which may appear abnormal should be reviewed. When muscle spasm is marked, deep palpation is painful, uninformative and unnecessary.

Persistent localized tenderness is the most important sign of inflammation. If it cannot be determined for sure that tenderness, *muscle spasm or other important signs actually exist, the examination* should be repeated after a short interval. It should be remembered that apprehension, suggestion, fatigue, exposure or lack of co-operation may give false positive or negative responses. When equivocal physical findings are reproducible on successive examinations or when they are verified by competent observers, they can be considered significant.

Percussion of the abdomen is helpful in demonstrating gas or fluid in the hollow organs or in the free peritoneal cavity. Gas in the bowel under pressure gives a high-pitched percussion note. When the abdomen is enlarged and resonant, consider intestinal distention or pneumoperitoneum. In pneumoperitoneum the area of normal liver

when in reality intestinal obstruction does exist. In acute appendicitis, constipation due to early inhibition of the intestinal motility may be expected. It may be associated with a sensation of cecal "gas stoppage" which is not relieved by enemas. Less commonly, diarrhea occurs with the onset of appendicitis, or may appear later when peritonitis develops. Diarrhea is often a prominent symptom in "food poisoning" or acute gastroenteritis. Bouts of alternating diarrhea and constipation suggest diverticulitis of the sigmoid or possible large bowel cancer.

BLEEDING.—"Tarry" or black stools generally indicate upper gastrointestinal hemorrhage. Rapid bleeding from any part of the gastrointestinal tract leads to the passage of liquid or clotted blood and signs of exsanguination. The mixed blood and mucus stools ("currant jelly") of intussusception of infancy are quite typical.

OTHER SYMPTOMS.—All of the patient's symptoms should be considered and logically and systematically analyzed with respect to the organs most likely to give rise to acute conditions. These conditions arise most commonly in the appendix, gallbladder, stomach and duodenum, pancreas, spleen, liver, small intestine, large intestine, the urinary tract and the female reproductive organs. An understanding of the structure and function, innervation, peritoneal covering and anatomic relationships of these organs will aid the physician in his interpretation of symptoms and signs to the end that a correct diagnosis can be established.

PHYSICAL EXAMINATION

The general physical examination provides essential data for making the diagnosis, determining the urgency of the condition, assessing the patient's physical state and operative risk, detecting imbalances and preoperative needs, timing the operation, selecting anesthesia and making a prognosis

The abdominal examination (including pelvic and rectal) provides information which indicates the type and degree of the intra-abdominal reaction upon which the differential diagnosis or direct diagnosis can be based and the need for or against operation.

The patient is surveyed rapidly for fever, shock, hemorrhage, anemia, dehydration, cardiac decompensation, etc. If necessary, essential treatment is started immediately, and the detailed history and examination are deferred temporarily.

The severity and character of the pain may be apparent. The

temperature, pulse, respiration and blood pressure are recorded; this information provides a base line for later observations. Careful examination of the heart and lungs should be carried out, bearing in mind that disturbances in these organs may mimic acute abdominal diseases. At this time also, the functional capacity of these organs is assessed. If there is need for special laboratory tests, x-rays, electrocardiograms or consultations, the machinery for securing them should be set in motion.

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dulness becomes resonant. Gastric distention produces an area of increased resonance in the left upper quadrant or sometimes in most of the upper abdomen.

Free fluid within the peritoneal space is demonstrated by testing for a fluid wave and shifting dulness. In ascites, bulging in the flanks may be observed. Small accumulations of peritoneal fluid often cannot be demonstrated by physical signs alone.

Auscultation is most helpful for assessing the functional activity of the bowel. Noises originating within the abdomen are produced by peristaltic movements transporting the bowel contents. The character of the bowel sounds is dependent on the thickness of the wall, the size of the lumen, the content of the bowel, the intraluminal pressure and the duration, frequency and vigor of the waves. Bowel sounds frequently are audible without the aid of a stethoscope (borborygmi).

Interpretation of bowel sounds can be learned only by practice and experience. Normally they occur as gurgles, rustles and rushes. They are influenced by digestion as well as by the emotional state. When alterations in the bowel sounds (absent, hypoactive or hyperactive) occur in association with other changes, they have clinical significance. Inhibition of gastrointestinal function is a part of the reaction to local and general stress. For example, peritoneal irritation, as well as an acute fracture of the femur, will cause a cessation of bowel activity and a silent abdomen. The inhibition generally does not persist; and after hours, or days, sounds will again be heard as function is resumed.

The auscultatory findings encountered in established mechanical intestinal obstruction may be striking. The sounds are loud, booming, rhythmical and synchronous with colicky pain. As the bowel becomes distended, the sounds become high pitched and take on a tinkling quality. The character of the bowel sounds is an important clue to the differential diagnosis of intestinal obstruction. In paralytic ileus the bowel sounds are absent or hypoactive. In mechanical obstruction they are hyperactive but later become hypoactive, as exhaustion, vascular impairment and distention-inhibition of the intestine develop.

The physical examination must include rectal palpation in the male and pelvic and rectal palpation in the female. Acute prostatitis and seminal vesiculitis in men may mimic acute intra-abdominal conditions. Fecal impaction, pelvic abscesses or neoplasms may produce signs of intestinal obstruction. When an inflamed appendix

lies low in the pelvis, there may be rectal tenderness and a palpable rectal mass in the absence of abdominal signs.

Some diseases of the female pelvic organs produce acute abdominal conditions. The pelvic examination may reveal a tubal or ovarian mass, enlargement of the uterus, exquisite tenderness on movement of the cervix, bulging in the cul-de-sac or a bloody or purulent cervical discharge. If any of these signs are encountered, acute pelvic complications must be considered.

Laboratory Examination

The urine and blood should be examined routinely. An analysis of a catheterized urine specimen is desirable in the female. The specific gravity and reaction of the urine is a rough index of the water and salt balance. Albuminuria suggests impaired renal function. Glycosuria in the absence of previous glucose infusion suggests diabetes. Ketonuria or acetonuria may indicate acidosis. Pus and blood in the urine suggest disease of the urinary tract (e.g., ureteral stone) but can also result from an inflamed appendix lying in proximity to the ureter or bladder.

In dehydration states, the red cell and hemoglobin values are increased as a result of hemoconcentration. Early in the course of simple intestinal obstruction there may be no significant alterations in the blood picture. The total leukocyte count and the percentage of polymorphonuclear cells and immature forms are usually elevated in acute inflammatory conditions. Conditions in which tissue necrosis occurs (e.g., strangulated intestinal obstruction) are generally associated with a marked polymorphonuclear leukocytosis.

Other laboratory tests may be helpful. The erythrocyte sedimentation test is sometimes an aid in the differentiation of early acute appendicitis from pelvic inflammatory disease. The red cell fall is accelerated in conditions associated with infection and abscess formation. Smears and cultures of cervical and urethral exudates may reveal the presence of specific pathogens (e.g., gonococcus).

The serum amylase test is essential when the possibility of acute pancreatitis exists. This condition should be kept in mind in all patients with acute upper abdominal pain. Serum amylase values in excess of 150 units (Somogyi) are significant, but levels of 1,500–2,000 units or more are not unusual in the early stages (twenty-four to forty-eight hours) of severe acute pancreatitis. The serum amylase

level falls rapidly to normal after this short initial period. The serum lipase determination, which is preferred by some physicians, but which requires more laboratory time, remains elevated for longer periods. It is used less frequently than the serum amylase test.

X-ray Examination

Important information can be secured from x-ray studies. Final interpretation of the films is best based on combined study by the clinician and the radiologist in order that the whole problem can be considered.

Generally, in patients with acute conditions of the abdomen, the x-ray examination consists of two abdominal exposures (scout films): a supine anteroposterior film and an upright film. When the patient cannot be placed in the upright (sitting or standing) position, a lateral decubitus film is made with the patient lying on his side (usually left) and the x-ray beam directed horizontally through the abdomen. A chest film may also be obtained at this time if indicated.

Except under special circumstances, administration of contrast substances is contraindicated. A barium suspension given by mouth or by rectum is likely to leak into the peritoneal cavity when perforation or impending perforation exists. Likewise, when there is an impending bowel obstruction, it may be made complete by introduction of barium. When large-bowel obstruction is suspected, a barium enema is sometimes required to establish the diagnosis. A judiciously administered barium enema is an important diagnostic and therapeutic (reduction) measure in intussusception of infants and children. If urinary tract injury or disease is suspected, intravenous pyelography is a readily available and probably harmless diagnostic procedure.

Scout films of the abdomen can often provide immediate information which confirms the diagnosis, excludes certain diagnoses and suggests previously unconsidered diagnoses.

The films may reveal specific alterations which are associated with the following conditions:

Pneumoperitoneum.—Gas in either or both subdiaphragmatic areas in the upright film (or in the flank in the lateral decubitus film) is almost pathognomonic of perforation of a hollow viscus. Most commonly the gas is due to a ruptured peptic ulcer or traumatic perforation.

Distention.—When an abnormal amount of fluid and gas is noted

in the bowel and is associated with an increase in the diameter of the bowel, one must consider intestinal obstruction, either mechanical or functional in origin. Gas in the small bowel is normal in the infant and young child but abnormal later in life.

Foreign bodies.—Gallstones, urinary stones, fetal parts, calcified myomas, surgical sponges or instruments, shell fragments, etc., may be encountered.

Localized Collections of Fluid or Gas.—The presence of localized collections of fluid or gas may suggest intraperitoneal perforation and abscess formation (e.g., appendicitis, diverticulitis and carcinoma).

Enlargement or Displacement of Organs.—Retroperitoneal hemorrhage, subcapsular splenic hemorrhage, acute pancreatitis, hydronephrosis, etc., may produce significant radiographic alterations.

Diagnosis and Differential Diagnosis

Investigation and supportive treatment should proceed concurrently in most situations, and especially if a specific diagnosis is not immediately apparent or cannot be established. This may be the case when the initial pathologic process has been complicated by secondary changes (e.g., shock in acute pancreatitis). The clinical picture at any one time is influenced by many variables and often changes rapidly. A broad and overlapping classification which takes into account the changing clinical picture, the gravity of the pathophysiologic disturbance, the urgency of the therapeutic problem and the seriousness of prognosis follows:

Catastrophe.—This category includes those conditions characterized by *sudden severe and continuous* abdominal pain, moderate to extreme abdominal tenderness and muscle spasm, and shock or a shocklike state. There is extensive tissue damage and fluid loss from traumatic, chemical or vascular insult. Emergency treatment, usually including operation, is required. Without treatment, rapid and progressive deterioration of the patient's general physical state may be expected. Prognosis should be guarded.

Colic.—The onset of colic may be variable, but periodic pain due to smooth-muscle spasm and serious disturbances in gastrointestinal function are characteristic. Profound systemic reactions are generally not encountered in the early stages but become severe as the process advances. The need for treatment is urgent but is not so critical as in the preceding category. The prognosis is good in cases seen early.

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later, as inflammation develops, the pain becomes more constant and is associated with tenderness.

The need for immediate medical care when the clinical picture

TABLE 19.—SPECIFIC CONDITIONS ACCORDING TO CATEGORIES

	SURGICAL	NONSURGICAL
Catastrophe	Rupture of a hollow organ—spontaneous or traumatic (peptic ulcer, ectopic pregnancy, etc.) Rupture of a solid organ—usually traumatic (spleen, liver, kidney) Acute vascular occlusion (mesenteric accident, strangulating obstruction) Massive hemorrhage, peptic ulcer, carcinoma, esophageal varices, etc.	Acute pancreatitis Coronary thrombosis Dissecting aneurysm
Colic	Acute intestinal obstruction —small and large bowel Acute appendicitis (see also Inflammation, below)	Gastroenteritis Fecal impaction Biliary colic Renal colic
Inflammation	Acute appendicitis Acute cholecystitis Acute diverticulitis	Mesenteric lymphadenitis Regional enteritis Pelvic inflammatory disease Ruptured ovarian follicle Urinary tract infection Pneumonia and pleuritis Hepatitis Diabetic acidosis Lead poisoning Tabes dorsalis Porphyria

is that of an abdominal catastrophe is suggested by the label "Priority I" in Table 20. The diagnostic and therapeutic implications of this category should be apparent.

SOME CHARACTERISTICS OF COMMON ACUTE CONDITIONS OF THE ABDOMEN

ACUTE APPENDICITIS.—Pain begins in the epigastric or periumbilical area and in a few hours shifts to the right lower quadrant. Nausea

INFLAMMATION.—In acute inflammations of the abdomen (Fig. 97) the initial manifestations may not be severe, but pain and tenderness may increase and tend to become localized. Shock is absent until the late stages. Fever and leukocytosis are usual. The bowel sounds are hypoactive or absent. Tissue damage is likely to be extensive. Signs of infection may be prominent. Operative and nonoperative measures

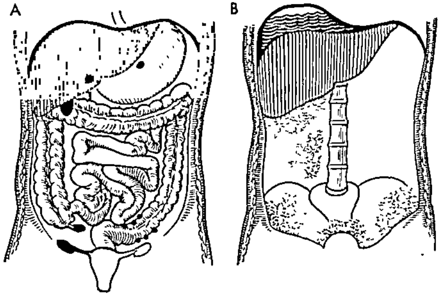

FIG 97.—Acute inflammations of the abdomen. *A*, common primary sites of

should be applied according to the diagnosis and indications. The prognosis is generally favorable.

The need for surgical and nonsurgical treatment is also indicated in Table 19.

Table 20 correlates the pathologic, clinical and therapeutic aspects of the most frequent acute conditions of the abdomen. It should be stressed that such a categorization is helpful only insofar as it is used with good judgment. All of the acute abdominal conditions are subject to great variations, and more than one mechanism is likely to be involved. For example, in acute appendicitis the initial pain is likely to be colicky as a result of obstruction of the appendical lumen;

and vomiting nearly always follow the pain rather than precede it. Localized right lower *quadrant tenderness is* the most significant single diagnostic sign. Muscle spasm and rebound tenderness are usually present. There is a slight fever and leukocytosis.

PERFORATED ULCER.—There may be a history of previous ulcer pain. The acute episode begins suddenly with upper and then diffuse abdominal pain. *The signs of peritonitis are severe, and there is true* boardlike rigidity. The tendency for free peritoneal fluid to gravitate down the right paracolic gutter to the right lower quadrant of the abdomen and localize an inflammatory mass may confuse the diagnosis. The upright film will reveal air under the diaphragm in about 85 per cent of cases.

ACUTE CHOLECYSTITIS.—In acute cholecystitis, there may be a history of indigestion and attacks of abdominal pain precipitated by ingestion of fatty foods. Nausea and vomiting are usual. The pain is located in the epigastrium and right upper quadrant and radiates to the back and the tip of the right scapula. There is tenderness and muscle spasm along the right costal margin, and the gallbladder is often palpable. A low-lying gallbladder or a high appendix may be a source of confusion. Gallstones are visible on the scout film of the abdomen (in about 20 per cent of cases) if the calculi contain sufficient calcium to make them radiopaque.

ACUTE INTESTINAL OBSTRUCTION.—A possible underlying etiologic factor is suggested by an abdominal scar, a hernia or a tumor. The organic obstructions are notable for *intermittent, cramping abdominal* pains which are synchronous with audible peristaltic rushes. Persistent vomiting, distention and constipation are characteristic. In uncomplicated obstruction, there is no fever. The scout films of the abdomen reveal dilated, gas- and fluid-filled loops of bowel.

RUPTURED ECTOPIC PREGNANCY.—The *important diagnostic points* in this condition are: sudden onset of lower abdominal or pelvic pain, *slight vaginal bleeding,* tenderness on movement of the cervix and a pelvic mass of varying consistency. There may have been a missed menstrual period, and the presumptive signs of pregnancy may be evident. Shock develops rapidly if the hemorrhage is *exsanguinating.*

ACUTE TORSION.—The pedicle of an ovarian tumor or cyst (*not a normal ovary*) *or a uterine* fibromyoma may become twisted and cause sudden severe abdominal pain, nausea and vomiting. The tumor may be difficult to palpate because of the extreme pain and muscle spasm.

TABLE 20.—CATEGORIES IN ACUTE CONDITIONS OF THE ABDOMEN *

PRIORITY	PATTERN	MECHANISM	CLINICAL PICTURE †	MANAGEMENT
I.	Pain, collapse, *shock* (catastrophic)—e.g., perforated ulcer, ruptured ectopic pregnancy, acute pancreatitis, mesenteric thrombosis, ruptured aneurysm, etc.	Perforation, hemorrhage, thrombosis, necrosis	Sudden severe pain, shock or shocklike state; abdominal tenderness; rigidity; silent abdomen; severe systemic reaction	Immediate resuscitative and supportive measures; diagnostic studies; early operation if indicated
II.	Pain (intermittent), *colic*—e.g., acute intestinal obstruction, biliary colic, ureteral colic, etc.	Obstruction of hollow muscular organ (smooth muscle); strangulation may be impending or existent	Recurrent cramping pains, vomiting; distention; noisy abdomen; systemic reaction, slight to moderate; x-rays may be diagnostic	Establish diagnosis if possible; correct systemic imbalances; early operation if indicated
III.	Pain, tenderness, *inflammation*—e g., acute appendicitis, acute cholecystitis, acute diverticulitis, acute salpingitis, etc.	Irritation due to bacterial, chemical, ischemic factors	Pain variable, usually increasing, tenderness—localized, then diffuse with rupture; muscle spasm, often a mass, systemic reaction—moderate to severe	Clinical diagnosis usually possible; early operation in appendicitis; proper timing of all therapy (fluids, antibiotics, operation)

* Suggested by Dr. C. C. Peterson.
† Generally there is a variable degree of gastrointestinal symptoms.

TABLE 21.—DIFFERENTIAL DIAGNOSIS OF UPPER ABDOMINAL PAIN *

	ACUTE APPENDICITIS	ACUTE CHOLECYSTITIS	PERFORATED PEPTIC ULCER	ACUTE PANCREATITIS	PLEURISY AND PNEUMONIA	CORONARY OCCLUSION
Age	Usually under 40	Over 40	30–50	30–50	Any age	Over 40
Sex	Both	Female, fat	Rare in females	Females predominate	Both	Male
Pain	Epigastric; shifts to R L Q., constant with exacerbations	Severe, radiates to back and shoulder, requires morphine, relieved by antispasmodic	History of ulcer in 60–75%, sudden onset, intense, constant pain, requires morphine	Sudden onset after large meal, severe, constant, radiates to back, requires morphine	In upper abdomen, not localized, relieved by splinting respiratory muscles	Lancinating; radiates to left shoulder and arm
Vomiting	Exception, but always anorexia	Reflex, may be much retching	Not prominent	Always	Exception	Reflex
Appearance	Not acutely ill until peritonitis	Worn because of pain	Acutely ill, keeps abdomen immobile; shocklike	Acutely ill, shocklike if necrosis	Restless; may have grunting respirations	Dyspneic; cyanotic; very restless; sweating, B P. subnormal
Temperature	99–100 F.; higher after perforation	99–102 F.	Subnormal	Subnormal at onset; later variable	100–103 F.	Normal to subnormal
Tenderness	Localized R.L.Q ; rebound	Localized R.U.Q.	Diffuse, more in upper abdomen; boardlike rigidity, absent bowel sounds	Epigastric, rebound; bowel sounds decreased	Epigastric, inconsistent; no rebound; no restriction of abdominal respiratory movement	Upper abdomen, but changeable and inconsistent
Laboratory	Leukocytosis usual	Leukocytosis	Leukocytosis	Serum amylase elevated; glycosuria occasional	High leukocytosis	Leukocytosis, ECG very helpful
X-ray	No help	May show stones or nonvisualization of gallbladder	Free air in 85% 4 hr. after onset	"Sentinel loop" of small bowel	Chest x-ray diagnostic	No help

* It should be emphasized that the data in this table represent usual findings and are not to be construed dogmatically. (From Requarth, W.: *Diagnosis of Acute Abdominal Pain* [Chicago: Year Book Publishers, Inc., 1953], p. 39.)

ACUTE PANCREATITIS.—There is sudden, usually severe, continuous pain across the upper abdomen and into the interscapular or upper lumbar region. There is associated nausea and vomiting, and often slight jaundice develops. Signs of shock may be striking. Tenderness is marked, but muscle spasm may be slight because the inflammation is largely confined to the lesser sac and retroperitoneal region. The serum amylase test is often diagnostic in the early stages.

ACUTE MESENTERIC THROMBOSIS.—Mesenteric occlusion can result from arterial thrombosis or embolism (usually from the heart). Venous mesenteric thrombosis may be spontaneous or secondary to regional inflammation. Severe diffuse abdominal pain is associated with nausea, vomiting, progressive distention and sometimes a bloody diarrhea. The signs of peritonitis develop rapidly. The white cell count is high. X-ray films may reveal widespread gas- and fluid-filled loops of bowel, but negative x-ray findings do not exclude this diagnosis.

RUPTURED OVARIAN FOLLICLE (MITTELSCHMERZ).—Rupture of a follicle with hemorrhage may produce acute abdominal pain. This condition occurs midway between the menstrual cycle and is characterized by lower abdominal pain which is usually not severe and which subsides within a few hours. Nausea and slight vomiting are common. Tenderness and muscle spasm are not striking. The temperature and leukocyte count are slightly elevated. Mittelschmerz often mimics appendicitis. The history and pelvic findings usually support the diagnosis, but a period of observation may be indicated.

ACUTE MESENTERIC LYMPHADENITIS.—This childhood condition often occurs with, or follows, an upper respiratory infection, and there may be other evidence of lymphadenitis. Lymphadenitis cannot always be differentiated from appendicitis. In both conditions, there is abdominal pain, nausea, vomiting, fever and leukocytosis. If appendicitis cannot be ruled out and there is no contraindication, operation is advisable.

REGIONAL ENTERITIS.—The acute phase of regional enteritis may mimic appendicitis. Abdominal pain is often mild and persistent. Vomiting is unusual, unless there is intestinal obstruction. The stools may be loose and frequent. There may be a palpable mass in the right lower quadrant of the abdomen.

RENAL AND URETERAL CALCULI.—Colicky pain is noted in the back or flank and in the lower abdomen and is referred into the thigh (never below the knee) and the external genitalia. Scout films of the lower abdomen may reveal a stone or enlarged kidney. There will

be hematuria and/or pyuria unless urinary obstruction or anuria exist.

Acute Salpingitis.—Symptoms most often appear during the premenstrual phase of the cycle. There may be a history of venereal exposure and vaginal discharge. Movement of the cervix produces severe abdominal pain if there is pyosalpinx. Pelvic tenderness is often bilateral, and there may be palpable adnexal changes. The erythrocyte sedimentation rate is usually high. Urethral and cervical smears may reveal Neisseria gonorrhoeae.

For a correlation of the outstanding factors in the differential diagnosis of upper abdominal pain, see Table 21; of lower abdominal pain, Table 22.

Treatment

Severe abdominal pain which persists for six hours or longer in a previously well patient generally indicates the need for surgical treatment. The common denominator of the acute surgical conditions of the abdomen is pain. Although the key to successful management lies in diagnosis, this does not mean that a specific etiologic diagnosis is possible in each case. Care must be taken to avoid unnecessary, harmful and ill-timed operations. Indicated operations on poor-risk or depleted patients must not be delayed, but every effort must be made to operate when conditions are optimal for recovery. There is a calculated risk in any operation, and it is generally increased under emergency conditions.

Preoperative Measures

In the *catastrophe group,* resuscitation is the major preoperative consideration. In shock, matched blood should be secured and transfusion begun as soon as possible. In the meantime, plasma expanders may be used. The stomach should be emptied by suction. Oxygen may be required. If hypotension persists at dangerous levels after adequate blood replacement (usually four units or more), a vasopressor drug may be indicated. The danger in the use of these drugs lies in depending on them to correct hypotension when blood replacement has been inadequate.

In the *colic group,* there is more time for studies and preparation. Patients with obstruction require correction of fluid-electrolyte imbalances. Transfusions may be necessary. The stomach and upper bowel should be emptied by suction. The long intestinal tube may be

TABLE 22.—Differential Diagnosis of Lower Abdominal Pain *

	Acute Appendicitis	Ureteral Obstruction	Acute Salpingitis	Ectopic Pregnancy	Diverticulitis
Age	Usually under 40	Under 40	Under 40	Under 40	Over 40
Sex	Both	Both	Female	Female	Male
Pain	Epigastric; shifts to R L Q ; constant with exacerbations	Severe, knifelike; begins in lumbar area, radiates to groin, scrotum, thigh; dysuria, frequency	Dull, constant, both L Q , recurrent attacks, jarring is painful, backache; dysuria	Sharp, knifelike	Dull cramping; L.L.Q. pain, diarrhea ±
Menses	—	—	No change or menorrhagia	Missed or scanty period, 15–25% have no irregularity	—
Temperature	99–100 F. before perforation	Normal	99–102 F.	Normal	99–101 F.
Tenderness	*Localized* R L.Q , rebound	*Costovertebral, none in* abdomen	*Bilateral* L Q.; suprapubic; rebound	*Unilateral* L Q , rebound	L L.Q ; rebound, mass ±, mild distention ±
Pelvic examination	Tenderness high on right	—	Exquisite tenderness on movement of cervix, profuse purulent discharge	Cervix moderately tender to movement, bloody discharge (dirty brown)	—
Laboratory	Hematuria 6%, normal sedimentation rate, leukocytosis	Hematuria, no leukocytosis	Skene and Bartholin positive for gonococcus, sedimentation rate elevated	Aschheim-Zondek may or may not be positive, cul-de-sac puncture—blood	Leukocytosis
X-ray	No help	See stone on flat plate 85%, I.V. pyelogram helps	No help	No help	No help unless barium x-ray previously showed diverticulosis

* *It should be emphasized that the data in this table represent usual findings and are not to be construed dogmatically.* (From Requarth, W.: *Diagnosis of Acute Abdominal Pain* [Chicago: Year Book Publishers, Inc., 1953], p. 37.)

operation will be a first-stage procedure, as in large-bowel obstruction due to cancer, when a decompressing colostomy may be established so that an extensive resection can be done later.

Care and closure of the abdominal incision constitutes an important part of the operation. *If wound healing is complicated by* hemorrhage, infection or unusual stresses, disruption and evisceration often follow. Wound complications are most likely to occur after emergency operations, when the procedure has been undertaken and completed in haste.

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introduced, but effective decompression of the small bowel cannot be expected until the tube has passed the pyloric barrier. Operation cannot be deferred if there is an established obstruction or if intestinal strangulation is a possibility. Hence, the introduction of the long tube should not be followed by protracted efforts to secure small-bowel intubation at the cost of delaying the required operation.

In the *inflammatory group*, broad-spectrum antibiotic therapy can be instituted preoperatively; but in most cases it is started after operation. Nasogastric suction and rest of the gastrointestinal tract (nothing by mouth) is generally indicated. Preoperative enemas are unnecessary and may be harmful. Fluids, electrolytes and blood should be supplied as indicated. As a rule, narcotics should be withheld until a working diagnosis and a plan of treatment has been established.

Operative Care

The patient should be transported to the operating room after he has been prepared and his condition is as nearly optimal as the circumstances permit. The preanesthetic medication will have been administered, and the anesthetic agent and technique selected. Anesthesia is induced, the operative field is prepared, and sterile drapes applied.

The location and type of abdominal incision will depend on the diagnosis as well as on the personal preference of the operator. Operative exposure must be sufficient to carry out the procedure without injury to other structures. This usually means that the operation must be performed under direct vision. It goes without saying that good lighting, capable assistants and proper instruments are needed.

The operation should be performed without haste, except in rare instances when the occasion demands haste. All the important principles of wound management should be observed.

The extent of the operation will vary according to the indications, the physical status of the patient, the local conditions in the abdomen and the experience and judgment of the operator. Often the operation will be simple, quickly accomplished and relatively atraumatic, as in appendectomy, closure of perforated ulcer or division of an obstructing peritoneal adhesion. At other times, it will be more complicated, as in gastric resection for massive bleeding from peptic ulcer, in cholecystectomy for acute gangrenous cholecystitis, or in bowel resection and anastomosis for strangulating obstruction. Occasionally the emergency

A "complete" hernia is an inguinal hernia which descends into the scrotum; an "*incomplete*" *hernia is one which descends in the inguinal* canal or through the external ring but does not enter the scrotum.

A Richter's hernia is one in which only a portion of the bowel wall is caught or "nipped" in the hernial ring, producing only partial occlusion to the bowel lumen. It may occur in any type of hernia but is most commonly found in the femoral hernias.

A *Maydl's hernia is a strangulation of the bowel in W form*: an inwardly directed loop of bowel is strangulated between two outwardly directed loops which occupy the sac.

A "sliding" hernia is one in which a part of the hernial sac is formed by a retroperitoneal organ, most often the cecum, descending colon, sigmoid or bladder. The organ which "slides" and the blood vessels supplying it become part of the sac wall. If this situation is unrecognized at operation, inadvertent but serious damage to the blood supply to the viscus may occur. Sliding hernias are most often of the inguinal type, either indirect or direct; but anatomically similar conditions are sometimes found in hiatal (diaphragmatic) hernia and complete rectal prolapse.

GENERAL FACTORS REGARDING ABDOMINAL HERNIAS

Genesis of Hernias

1. Hernias may result from a chronic sustained increase in intra-abdominal pressure from any cause: (*a*) any space-occupying lesion or enlargement within the abdomen (tumors, cysts, pregnancy, ascites, etc.); (*b*) *transmitted pressure from coughing, straining, vomiting* (prostatism, chronic constipation or chronic pulmonary infections); or (*c*) a gravitational effect, which aids the descent of *hernial contents into the dependently located inguinal or femoral hernial sac.*

2. Hernias may result from a congenital or acquired weakness or deficiency of the abdominal wall. (*a*) Direct inguinal hernia develops in a relatively unprotected area of the abdominal wall (Hesselbach's triangle). There may be a relative weakness of the transversalis fascia or a high insertion of the conjoined tendon into the rectus sheath. (*b*) The deficiency may be a muscle atrophy, secondary to nerve injury. For example, injury to the iliohypogastric and ilio-inguinal nerves during appendectomy through a McBurney incision may be followed by the development of a right indirect inguinal hernia. (*c*) The deficiency may be incompetence of the inguinal

CHAPTER 22

Abdominal Hernia

AN ABDOMINAL hernia is a congenital or acquired protrusion of peritoneum through a defect in the abdominal wall. A hernia consists of three parts: a defect in the abdominal wall, a sac and the contents of the sac. Hernias are generally classified according to their anatomic location. The more common types of hernia include:

1. Inguinal, indirect and direct
2. Femoral
3. Umbilical
4. Epigastric
5. Incisional or postoperative ventral
6. Diaphragmatic
7. Rarer forms: internal (retroperitoneal, intraperitoneal), obturator; lumbar; sciatic; perineal

The relationship of the contents to the sac forms a basis for classification, as follows:

1. *Reducible.*—The contents of the sac may be returned to the peritoneal cavity.
2. *Irreducible or Incarcerated.*—The contents have become adherent to the sac and cannot be returned to the abdomen. Neither obstruction nor vascular interference has occurred.
3. *Strangulated.*—There is interference to the circulation of the sac contents, usually the bowel. Intestinal obstruction and strangulation are most frequently associated.

A congenital hernia is due to a developmental defect. Although the hernia may not be apparent for many years, the underlying embryologic fault can usually be demonstrated at operation. An acquired hernia has no demonstrable developmental defect but results from weakness or separation of supporting tissues. Both congenital and traumatic factors may be associated in the development of a hernia.

is an abrupt elevation in intra-abdominal tension due to severe muscular exertion produced by heavy lifting, a fall or compression of the abdomen.

4. A preformed sac, derived from the unobliterated portion of the processus vaginalis which communicates with the abdominal

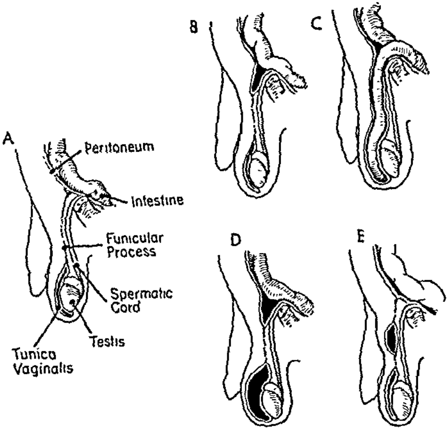
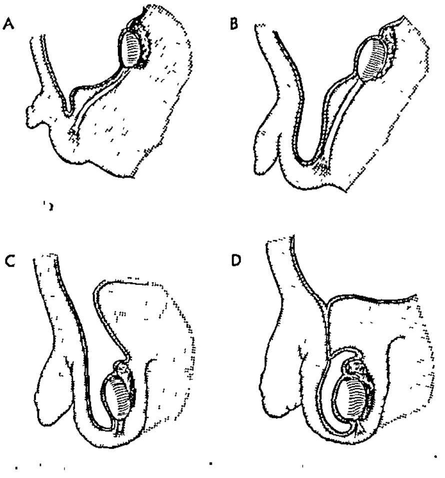


FIG. 99.—The basis for indirect inguinal hernia in the male. *A*, normal anatomic relationships. Note the obliterated funicular process and the tunica vaginalis testis. *B*, incomplete indirect inguinal hernia. C, complete (or scrotal) indirect inguinal hernia. The peritoneal pouch remains open throughout its entire extent. *D*, incomplete indirect inguinal hernia and hydrocele of tunica vaginalis testis, noncommunicating. *E*, hydrocele of the spermatic cord (incomplete obliteration of the funicular process) without hernia.

cavity, usually underlies the development of an indirect inguinal hernia. Normally, this process becomes obliterated and forms a fibrous cord except in its distal extremity, where it remains as the *tunica vaginalis testis* (Fig. 98). If obliteration is incomplete and a communication with the abdominal cavity remains, a potential or actual congenital hernia results. A sequestrum of the processus vaginalis may

"shutter." The internal ring is inadequately closed or supported when contraction of the conjoined tendon occurs. (*d*) The "weak spot" in the superior part of the umbilical ring may be the site of election for an umbilical hernia. (*e*) There may be maldevelopment of the embryo and a deficient closure of the abdominal wall, with failure of



remains as the tunica vaginalis testis.

the abdominal contents to return to the abdomen. This is called "hernia into the cord," or omphalocele. It is not a true hernia but an eventration of the abdominal wall. (*f*) When there is imperfect wound closure or defective wound healing, postoperative incisional hernia may develop.

3. The exciting cause of a suddenly "acquired" hernia generally

operative hernias), it is an important and a necessary precaution to establish the right of domain before undertaking operative repair. This may be tested by having the patient wear a support to retain the hernia for a period of days before operation.

COMMON ABDOMINAL HERNIAS

Inguinal Hernias

The inguinal canal, an oblique opening in the abdominal wall about 2 inches in length, contains the spermatic cord (round ligament in the female), the ilio-inguinal nerve and filaments of the genitofemoral nerve. The internal inguinal ring is a funnel-shaped aperture in the transversalis fascia, lying midway between the anterosuperior iliac spine and the pubic spine, about ½ inch above the inguinal ligament. The fibers of the transversalis fascia continue down over the cord as the internal spermatic fascia. The aponeurosis (conjoined tendon) of the internal oblique and the transversus abdominis muscles covers the internal ring and constitutes an effective "shutter" when the muscles of the abdominal wall are contracted. The external ring is a triangular opening in the aponeurosis of the external oblique muscles. The normal opening will admit the tip of the examining finger.

The walls of the inguinal canal are: anteriorly, the aponeurosis of the external oblique muscle; superiorly, the lower fibers of the internal oblique and the transversus abdominus muscles; posteriorly, the transversalis fascia; and inferiorly, the shelving border of the inguinal ligament.

The spermatic cord contains the vas deferens, the artery of the vas, the internal and external spermatic arteries, lymphatic channels draining into the lumbar nodes, the vaginal ligament and vestigial remnants of the funicular process. During its passage through the inguinal canal the spermatic cord acquires three coverings: the internal spermatic fascia from the transversalis fascia, the cremasteric fascia and muscle from the internal oblique muscle, and the external spermatic fascia from the external oblique muscle.

The sac of an indirect inguinal hernia lies in the spermatic cord, anterior to the vas deferens, and therefore passes through the internal ring and obliquely down the inguinal canal, and often out the external ring into the scrotum (Fig. 100). It lies lateral to the inferior epigastric artery.

also remain open and form, according to its location, a hydrocele of the cord or of the testicle (Fig. 99). A sac called the "femoral diverticulum" may similarly be the basis for a congenital femoral hernia.

Complications of Hernias

A hernia may become incarcerated, owing to adhesions binding the sac contents, inflammatory changes and narrowing of the neck of the sac, a sudden enlargement of the contents of the sac or loss of the "right of domain." The right of domain is said to be lost in large hernias (e.g., large inguinal or ventral hernias) when the capacity of the abdominal cavity is inadequate to accommodate the reduced hernial contents. Sudden reduction of large or long-standing hernias can produce serious alterations in intra-abdominal and intrathoracic pressure relationships, leading to cardiac and/or pulmonary insufficiency.

When bowel obstruction is due to hernia, there is always danger of strangulation of the blood supply. It may be impossible to ascertain, on clinical grounds alone, the presence or absence of such vascular impairment. The differentiation must usually be made at operation.

When strangulation exists, it tends to be a progressive process and, unless relieved, leads to tissue necrosis, perforation and peritonitis.

Preoperative Considerations

The repair of an abdominal hernia should be done as an operation of election unless the hernia is complicated and an emergency situation exists. Careful attention should be paid to the following matters:

1. The conditions are not optimal if the patient has a chronic cough. Not only should the question of the possible postoperative recurrence of the hernia be considered, but also the hazard of postoperative pulmonary complications.
2. There may be causes for increased intra-abdominal tension which require attention—e g., the straining incident to prostatic obstruction in men, phimosis in little boys, chronic constipation, etc.
3. Weight reduction is indicated if the patient is obese. In such a patient, wound healing is often complicated and the recurrence rate high.
4. If the hernia is large and difficult to reduce (especially post-

operative hernias), it is an important and a necessary precaution to establish the right of domain before undertaking operative repair. This may be tested by having the patient wear a support to retain the hernia for a period of days before operation.

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The anatomy of a direct inguinal hernia is quite different. It has nothing to do with the spermatic cord. Direct hernias are so named because the bulge takes the most direct route through the abdominal wall. The bulge or weakness originates in the Hesselbach triangle, the boundaries of which are: medially, the rectus sheath; laterally, the inferior epigastric artery; and inferiorly, the inguinal ligament. This

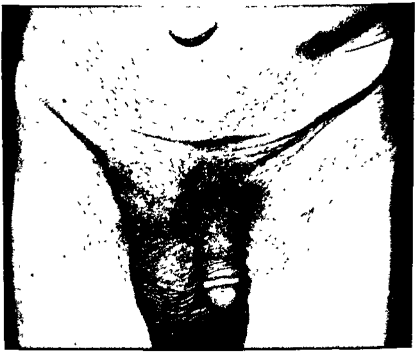

FIG. 100.—Large indirect inguinal (complete or scrotal) hernia and umbilical hernia.

area is supported by the transversalis fascia and the conjoined tendon (internal oblique and transversus abdominus muscles). Anterior to Hesselbach's area lies the external inguinal ring. If the conjoined tendon has a high insertion and the transversalis fascia is weak, Hesselbach's area is relatively unsupported and gives way under stress, resulting in a direct inguinal hernia, which often appears through the overlying external ring.

SYMPTOMS AND SIGNS.—There may be few, if any, symptoms associated with congenital indirect inguinal hernia of infants and children. When the hernia appears suddenly, following some unusual stress or strain in later life, slight pain and discomfort are the rule. The outstanding complaint is the presence of a bulge in the inguinal region. Often

the patient will observe that the mass disappears when he lies down. Characteristically, there is an inguinal swelling with an expansile impulse, which is transmitted to the examining finger when the patient coughs or strains. The mass may be easily reducible and nontender. The *examination for hernia should* be made with the patient in both the upright and the supine positions. It can be done without hurting the patient. It is usually possible to insert the index or little finger into

FIG. 101.—Groin hernias: their location with respect to the internal opening and the external bulge. *A*, indirect inguinal hernia. The sac passes through the internal [illegible] Direct hernias rarely enter the scrotum. *C*, femoral hernia. The sac passes through the femoral ring and presents below the inguinal ligament at the fossa ovalis.

the external ring to palpate the bulge; but if this cannot be done (e.g., in a child), the hernia can usually be demonstrated by external examination. An attempt should be made to differentiate between a direct and an indirect hernia. In indirect hernia (*Fig. 101, A*), the point of origin of the sac is the internal ring. When the hernia is reduced, external pressure applied with the index finger, pressing on the region of the internal ring, will prevent the appearance of the bulge. Direct hernias originate in Hesselbach's triangle, which lies directly behind the external ring. If the finger can be introduced

through the external ring, it will pass directly into this posterior defect. Pressure over the external ring will contain the bulge of a direct hernia, but pressure over the internal ring will not. Differentiation may sometimes be made in this manner.

An indirect and a direct hernia may exist concomitantly, forming a combined, or "pantaloon," type of inguinal hernia.

Direct inguinal hernias (Fig. 101, *B*) are rare in women and children and most common in elderly men. They are often bilateral. The sac is usually broad based and oval or dome shaped, and occasionally it descends into the scrotum. Direct hernias are generally easily reducible, and strangulation is uncommon.

Differential Diagnosis of Inguinal Masses.—The following conditions should be considered in the differential diagnosis:

Hydrocele.—There may be a hydrocele of the tunica vaginalis testis or a hydrocele of unobliterated portions of the vaginal process in the spermatic cord. The mass is not reducible, and there is no palpable impulse on cough. Because a hydrocele contains clear fluid, the transillumination test is positive. This test is best done in a darkened room with a flashlight or a special light.

Indirect inguinal hernia and hydrocele are frequently associated. In congenital hydrocele of infants and children, a hernia can be demonstrated in most instances.

Inguinal Adenitis.—A discrete, enlarged lymph node in the groin may be confused with a hernia. Nodes are often tender and inflamed. The site of an infection producing an enlarged lymph node in the groin may be the skin of the legs or feet, the genitals or the anorectal area. Such a node will be irreducible and may become suppurative.

Undescended Testicle.—In cryptorchism (undescended testicle), one or both testicles may be absent from the scrotum. Ectopic testicles in the inguinal canal or groin are palpable. Occasionally they are located within the abdomen. Cryptorchism is almost always associated with an indirect inguinal hernial sac.

Femoral Hernia.—The diagnosis of femoral hernia (Fig. 101, *C*, and 103) may be missed if the hernial sac which presents at the fossa ovalis is reflected up and over the inguinal ligament. Femoral hernias are sometimes irreducible. The location and point of origin is below the inguinal ligament and lateral to the pubic spine. In uncomplicated femoral hernias, no defects exist in the inguinal region.

Treatment of Uncomplicated Inguinal Hernia.—In addition

to surgical repair, trusses and the injection treatment may be considered.

1. Although infants are often fitted with yarn trusses in the hope of obliterating the patent processus vaginalis, *it is unlikely that anything short of operative repair will effect a cure.*

2. A properly fitted truss may be used for patients who are poor surgical risks and for those who refuse operation. Some of these patients will later have to have an emergency operation for strangulation, often when conditions are most unfavorable. Elective repair is therefore advisable in most instances.

3. The injection treatment of hernia, using various sclerosing solutions, *has been discarded* because of the high recurrence and complication rates.

4. Operative repair is the treatment of choice.

Operative Technics.—The principles of surgical repair of an inguinal hernia comprise: (*a*) high ligation and removal of the hernial sac and (*b*) reconstruction of the inguinal canal.

The standard types of surgical repair of an indirect inguinal hernia differ chiefly with regard to the disposition of the spermatic cord. The following are the four common types of repair:

1. *Bassini.*—The spermatic cord is placed above the conjoined tendon and below the external oblique aponeurosis. The conjoined tendon, therefore, is sutured to the inguinal ligament beneath the cord.
2. *Ferguson.*—The spermatic cord is placed between the conjoined tendon and the transversalis fascia, in what is essentially its normal position.
3. *Halsted.*—The spermatic cord is placed in the subcutaneous tissue above the aponeurosis of the external oblique muscle. A new internal ring is fashioned in the aponeurosis.
4. *Cooper's Ligament Repair.*—In this relatively new type of repair, the transversalis fascia is sutured to the superior pubic ligament (Cooper's) rather than to the inguinal ligament. This method is most commonly used for the repair of recurrent inguinal hernias, combined indirect-direct hernias and femoral hernias. The position of the spermatic cord is not altered.

In the infant with an indirect hernia, repair commonly consists of simple ligation and removal of the sac. It is usually unnecessary to disturb the cord or to suture the structures of the inguinal canal. Great

care must be taken to avoid injury to the inguinal nerves, the spermatic cord and the testicles. When the sac is "complete" (into the scrotum), it should be divided in its midportion and only the proximal portion removed. The distal portion, which remains in situ, then serves its normal function as the tunica vaginalis testis. Bilateral congenital hernias are common and are often repaired at one operation.

In the adult, high removal and ligation of the sac with repair of the defect in the inguinal canal is necessary. Before the sac is closed, the finger should be introduced through the internal ring into the abdominal cavity, to demonstrate positively the presence or absence of associated groin hernias in Hesselbach's area or the femoral canal. If present, these must also be repaired.

Successful hernia repair requires that the tenets of Halsted be followed to the letter. Infection, hematoma formation, excessive amounts of retained irritant foreign material (sutures, devitalized tissue), tension and improper repair are the common causes of recurrence.

The same principles apply to the repair of a direct hernia. Because the sac is often a wide bulge rather than a narrow-necked pouch, it is often not excised, but rather obliterated by overlapping or imbrication. Closure of the existing defect in the transversalis fascia constitutes the single most important step in the repair. Secure closure demands that fibrous tissue be sutured to fibrous tissue (fascia to fascia). When the defect is large, an autogenous graft from the external oblique aponeurosis or the fascia lata of the thigh may be required to close the defect. Occasionally, foreign substances such as tantalum or stainless steel mesh may be required.

Incarcerated Hernia

An incarcerated hernia may be replaced by manual reduction, termed "taxis." Taxis is potentially dangerous. The maneuver is contraindicated when the patient cannot be kept under close observation following manipulation and whenever the possibility of strangulation exists. The strict rules which should govern the manual reduction of hernias are: (1) apply minimal, sustained, nontraumatizing pressure; and (2) do not persist or force reduction if difficulty is encountered. The great dangers of taxis are. (1) the possibility of returning strangulated or injured hernial contents into the abdominal cavity, (2) reduction *en masse* (*i.e.*, the contents of the sac, along with the con-

stricting ring of peritoneum, are returned into the abdomen and obstruction or strangulation persists) and (3) injury to viscera in the sac.

The following points may be of value in the manual reduction of an incarcerated hernia: (1) *In most instances, gentle pressure over the hernia, with the patient in the supine position, the thighs flexed and the foot of the bed elevated, will suffice.* (2) *If the patient is a child who is crying or restless, it is helpful to give a sedative or analgesic to produce muscle relaxation.* (3) Cold applications (e.g., an ice bag) may help reduce edema and facilitate reduction.

Strangulated Hernia

Any hernia which suddenly becomes incarcerated and is not easily reducible, or has been "out" for a number of hours, is best treated by immediate operation. Frequently, manipulation simply makes the incarceration more severe. Continuous pain or intermittent colic suggests that obstruction and vascular strangulation are imminent or established. Delay under these conditions simply decreases the patient's chances for recovery. *The safe policy, and the only logical treatment, is operative exposure, inspection, reduction of the bowel (or resection, if necessary) and repair of the hernia.*

Sliding Hernia

The sliding hernia (Fig. 102) is a complication of the inguinal hernia in which a retroperitoneal structure (descending colon, sigmoid, bladder or cecum) enters the inguinal canal or the scrotum but lies outside the peritoneal hernial sac rather than within it. The relationship of the retroperitoneal structure to the hernial sac is similar to the relationship between the testicle and the funicular process during the descent of the testicle; i e., the testicle and its blood supply lie outside the funicular process (sac) but apposed to it—actually, the testicle forms a part of the sac wall. The testicle cannot easily be separated from the normal sac (tunica vaginalis testis) without injuring the testicle or its blood supply. Similarly in sliding hernia, the abdominal organ is attached to the wall of the hernial sac. The sac itself may contain small intestine or omentum, as does the sac of an uncomplicated or simple hernia.

At operation it is necessary to recognize the condition in order to

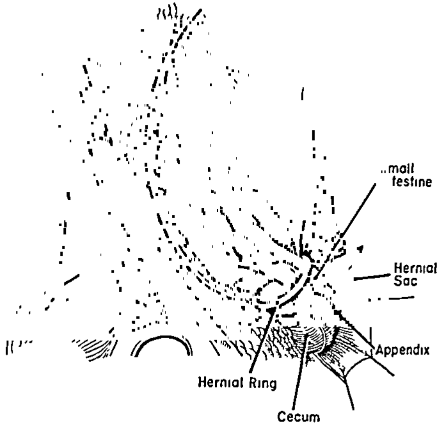

FIG. 102.—Sliding hernia of the cecum. Note that the anterior wall of the cecum forms the posterior wall of the hernial sac. The sac contains small intestine.

avoid injury to the bowel or its blood supply. The contained viscus is replaced in the abdomen by gradually separating it from below, or by reducing it through a separate abdominal incision. Then the neck of the hernial sac is closed, the sac removed, and the defect in wall repaired in the usual manner.

FEMORAL HERNIA

Femoral hernias constitute about 4 per cent of all hernias. They are more common in women than in men, and they rarely occur in children. However, inguinal hernias are more frequent in women than femoral hernias. While inguinal hernias are about eight times more common than femoral hernias, the incidence of strangulation is about equal.

The femoral canal, which is the most medial compartment of the femoral sheath, is normally filled with areolar tissue. The boundaries

of the femoral ring are: the inguinal ligament (anterior); the lacunar ligament (medial); the femoral vein (lateral); and the superior pubic fascia, or Cooper's ligament (posterior).

SYMPTOMS AND SIGNS.—The presence of a mass over the thigh in the femoral area, which is reducible and transmits an impulse on cough, is the usual picture of a femoral hernia (Fig. 103). The hernia appears through the femoral ring (Fig. 101, *C*), but occasionally the sac is reflected over the inguinal ligament, thus making differentiation

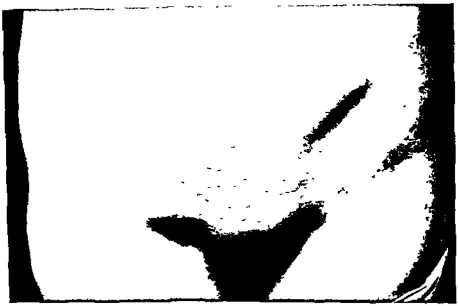

FIG. 103.—Femoral hernia.

from inguinal hernia difficult. This problem can often be resolved by noting that the origin of the sac is lateral to the pubic tubercle and medial to the femoral vessels. Because of the relative rigidity of the femoral ring, hernias through it are prone to become strangulated. Sometimes only a slight bulge may be noted in the femoral ring when symptoms of intestinal obstruction are unequivocal. In all cases of intestinal obstruction of obscure origin, a careful search for hernia should be made in both the femoral and inguinal regions. The small neck of the sac may render the hernia irreducible, and under these conditions there may be no detectable impulse on cough. The femoral ring is the most frequent site of Richter's hernia, in which only a portion of the wall of the intestine is held in the sac and the lumen of the bowel may be either partially or completely blocked.

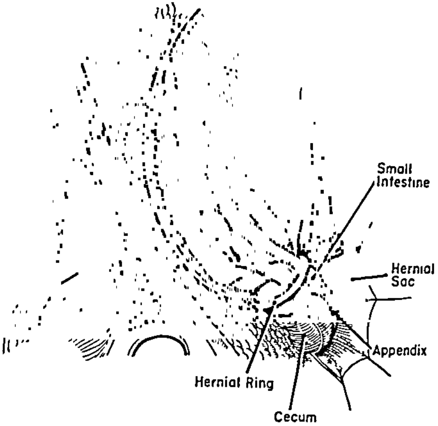

FIG. 102.—Sliding hernia of the cecum. Note that the anterior wall of the cecum forms the posterior wall of the hernial sac. The sac contains small intestine.

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repair of the omphalocele is carried out later, when the child has grown and the abdominal cavity has enlarged to the point where it can receive the displaced organs.

The infantile type of hernia occurs early in life and is due to improper closure of the umbilical ring. In the adult, umbilical hernia usually results from prolonged increased intra-abdominal tension. The hernia appears through a "weak spot" in the superior part of the ring. It may develop during pregnancy and is often associated with obesity. The hernia may be small and symptomatic, or very large and silent.

The signs of an umbilical hernia are: (1) protrusion of a mass at the umbilicus, usually reducible when the patient is lying down; (2) an impulse on coughing; (3) often, if there is intestine in the sac, a "gurgling" noise when the hernia is reduced; (4) generally easily defined hernial ring margins; and (5) sometimes incarceration and strangulation of the bowel or omentum, especially when the neck of the sac is small. As a rule, hernias with large openings are less likely to become incarcerated than those with small openings.

Umbilical hernia may be treated as follows:

1. In "hernia into the cord" (omphalocele), which is not common, repair or immediate covering of the sac and viscera with skin from the abdominal wall is necessary.

2. An infantile umbilical hernia can be retained but is rarely cured with repeated application of wide adhesive tape strapping. The tape should be applied so that there is an overlapping of the margins of the umbilical ring. The skin should be prepared with compound tincture of benzoin before the tape is applied. The strapping must be changed at least weekly. Elastic adhesive tape is preferred to plain tape. The mother can be taught to apply the support.

3. The surgical repair of an umbilical hernia consists in dissection and removal of the sac, followed by repair of the defect. This is accomplished by overlapping the fascia, usually in a transverse manner, referred to as the "vest-over-pants," or imbrication, technic. In children particularly, the umbilicus should always be preserved in order to avoid psychic trauma, which may follow its removal.

Epigastric Hernia

In epigastric hernia, there is a bulge in the midline above the umbilicus. Such herniations originate through small defects in the fascia of the linea alba. A peritoneal sac is present in most instances,

Differential Diagnosis of Femoral Masses.—In the differential diagnosis for femoral hernia, the following should be considered:

Inguinal Hernia.—If the femoral sac passes over the inguinal ligament, it may be confused with an inguinal hernia. It may be possible to define the femoral aperture when the hernia is reduced.

Saphenous Varix.—This condition is usually associated with varicose veins. When the patient coughs, a thrill (rather than an impulse) is transmitted down the saphenous vein. The varix usually lies lateral to the femoral ring. The pressure in a varix promptly falls when the patient lies down.

Lymphadenitis.—In lymphadenitis there may be several enlarged, tender inguinal nodes. Bilateral involvement is common. No impulse will be present on cough. A focus of infection in the foot, leg, groin or perineal area is usually present.

Psoas Abscess.—In this condition the mass in the groin is usually fluctuant and cannot be reduced, and there is psoas spasm with evidence of vertebral disease. Most often it is of tuberculous origin.

Treatment of Femoral Hernia.—Trusses are rarely satisfactory in the treatment of femoral hernia because the reduction cannot be maintained. Operative repair is required for cure. Generally, an inguinal incision is preferred to a femoral incision directly over the hernia. The sac is ligated and removed. The inguinal ligament is united with the pectineal, or Cooper's ligament with interrupted sutures. The transversalis fascia is closed, and the remainder of the repair is similar to the operation for inguinal hernia.

Umbilical Hernia

In umbilical hernia, there may be (1) a congenital hernia into the cord (omphalocele) or more commonly (2) a hernia through the umbilical ring in an infant or adult.

Omphalocele is not a true hernia but an eventration of the abdominal wall. The defect is present at birth. The covering of the sac is very thin (amnion and a fine layer of parietal peritoneum), and the abdominal viscera are visible through it. There is imminent danger of rupture of the sac, with evisceration and peritonitis. The organs (liver, bowel, stomach, etc.) within the sac usually cannot be replaced within the small abdominal cavity, and covering for the omphalocele must be secured immediately. This is accomplished by shifting skin flaps and allowing the herniation to remain temporarily. Definitive

to follow such complications as wound infection or disruption. They are almost certain to develop after wound disruption unless proper secondary closure is accomplished. The local and general factors influencing wound healing (detailed in Chapter 2) provide the basis for averting the development of this complication.

For the prophylaxis of incisional hernia, clean atraumatic surgical technics, careful closure of the abdominal wound and optimum conditions for healing are required. The importance of preventing postoperative distention, vomiting and excessive coughing cannot be overemphasized.

Small incisional hernias can sometimes be adequately controlled with an abdominal girdle or supporter. Some hernias will enlarge despite adequate support; these require surgical repair.

Surgical repair is accomplished by dissecting out the sac and approximating or overlapping the fascial layers of the abdomen. Weight reduction is indicated for the obese patient. In large hernias, the "right of domain" must be established.

Diaphragmatic Hernia

In this, less common, type of hernia there is a protrusion of the abdominal viscera into the thoracic cavity. The defect in the diaphragm may be congenital, acquired or traumatic in origin.

Congenital hernias result from a failure of closure of the pleuroperitoneal hiatus (Bochdalek), which is located in the left leaf of the diaphragm. There may be no hernial sac, and serious symptoms are usually present at birth or develop soon thereafter. Emergency treatment is required. Acquired diaphragmatic hernias frequently occur through the esophageal hiatus. Many are small and asymptomatic. Traumatic rupture or herniations of the diaphragm are produced indirectly by crushing injuries to the chest or abdomen which cause sudden severe changes in pressure relationships, or directly from lacerations (e g., stab wounds, bullet wounds).

The outstanding symptoms of diaphragmatic hernia are those resulting from embarrassment of cardiorespiratory and gastrointestinal functions, including reduction of ventilation and intestinal obstruction. The diagnosis is usually established by x-ray studies of the chest. Surgical repair is required for congenital and traumatic herniations of the diaphragm and for selected cases of hiatus hernia.

although sometimes only a conglomeration of properitoneal fat resembling a lipoma is found. An impulse on cough may not be demonstrable if the mass is irreducible.

Epigastric hernias generally cause only minimal symptoms, such as slight tenderness or pain. Often symptoms arising from an existing peptic ulcer, gastric cancer or gallbladder disease are attributed by the patient to a long-standing epigastric hernia. One must not be mis-

FIG. 104.—Giant postoperative ventral hernia, following wound disruption, which has "lost the right of domain," that is, the contents of the hernial sac could not be reduced without seriously impairing cardiorespiratory function. Successful surgical repair followed a preliminary course of induced pneumoperitoneum to gradually increase the capacity of the abdominal cavity.

led by the patient who believes all his distress comes from the hernia. To avoid this error, a detailed investigation is usually indicated before an operation is performed. The repair consists of removing the sac or fat tab and closing the fascial defect.

INCISIONAL AND VENTRAL HERNIA

This type of hernia follows a surgical operation or other trauma to the abdominal wall. It often occurs through vertical abdominal incisions; less frequently through muscle splitting (e.g., McBurney), transverse or oblique incisions. Postoperative hernias (Fig. 104) are likely

CHAPTER 23

Abdominal Injuries

Whatever the traumatic agency . . . laceration by spike or stake, a stab with a knife, dirk or dagger, bayonet thrust, or the more frequent gunshot wound or bullet wound, shell or bomb . . . the general principles of treatment are alike. A penetrating wound in the abdomen probably means a penetrating wound of the bowel or other abdominal viscus, and demands the earliest surgical intervention, unless a wisdom of prescience born of great experience justifies restraint.—GORDON-TAYLOR*.

IN PEACETIME, abdominal injuries most frequently are the result of automobile accidents, industrial and farm mishaps, sports involving body contact, falls and penetration produced by firearms, knives and ice picks. In warfare, they result from rifle or shell fragments, impingement, or crush and blast injuries, in addition to the forms of trauma mentioned above, and are characterized generally by more extensive parietal and visceral damage. The mortality from penetrating abdominal injuries is high, but a notable reduction has occurred since the early days of World War II, when it approximated 50 per cent. During the Korean conflict the mortality was about 10 per cent. This reduction has been made possible through the availability of early supportive and definitive surgical treatment, improvements in transportation of the injured, application of the many adjuncts to the surgical regimen (whole blood, improved anesthesia, antibiotics) and a better understanding of the principles and practices of operative repair and postoperative treatment.

Although civilian injuries are generally not comparable in degree or complexity to those sustained in warfare, the principles of treatment

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relatively fixed. It should be recalled that some portions of the gut (duodenum and ascending and descending colon) are in part intraperitoneal and in part extraperitoneal. Leakage from perforations of these organs can occur into the peritoneal cavity, into the retroperitoneal tissues or into both areas, depending on the location of the rupture. When rupture occurs on the extraperitoneal surfaces of these organs, the usual signs of peritoneal irritation will not appear and the diagnosis of perforation may be missed.

The intraperitoneal solid organs (liver and spleen) are relatively immobile and unusually soft and friable. They are easily contused, lacerated or ruptured. Hemorrhage may occur immediately or subsequently from rupture of a subcapsular hematoma. When the liver is lacerated, extravasation of bile also occurs. Rapid loss of circulating blood volume and shock, together with signs of peritoneal irritation, follow rupture of the solid organs. The bile ducts and pancreas are less frequently damaged in nonpenetrating injuries, but both are very serious. Rupture of the bladder produces extravasation of urine and blood into the extraperitoneal and/or intraperitoneal spaces according to the location of the rupture. Fractures of the pelvis, and especially of the ramus of the pubis, can cause bladder or urethral lacerations. Injury to the kidney usually results in extraperitoneal extravasation of blood and urine.

Penetrating wounds of the abdomen are most often gunshot or knife wounds, but occasionally they result from impalement and instrumentation (e.g., curettage of the uterus). Gunshot wounds usually produce injury to several organs and commonly traverse both the abdominal and thoracic cavities. To some degree, the internal injury can be gauged by determining the nature of the agent producing the penetration and the position of the patient when shot or stabbed, by the location of wounds of entrance and exit, by the presence of foreign bodies and of pneumoperitoneum or hemopneumothorax and by the fractures observed on the x-ray studies. Bullet wounds tend to follow a straight-line course through the body except when deflected by bone. The damage produced to the soft viscera is generally much greater than might be expected from the appearance of the external wounds. In knife wounds, the manner and force with which the knife is thrust, the length of the blade and the location of the wound of entrance will suggest the probable organ (or organs) damaged; but this can only be an educated guess. Introducing a probe into a stab or bullet wound to determine whether penetration has occurred is

are the same. It is safe to say that, even under the more nearly ideal conditions of peacetime practice, the management of patients with serious intra-abdominal injuries can be difficult. The chief difficulties encountered relate to recognition of the condition, shock, hemorrhage and infection. In nearly all abdominal injuries there is contusion of or penetration of the abdominal wall. The injury produces superficial or deep disruption of tissues, hemorrhage, extravasation of gastrointestinal fluids, vascular damage, infection, etc. In most abdominal injuries, the presence of visceral damage can be inferred from the history of the injury, the physical findings and the patient's clinical course, aided to some extent by blood, urine and x-ray findings. The problem, therefore, is quite different from that posed by injury to the extremities or the parietal structures; rather, it is similar to that encountered after head or thoracic injury.

Frequently the problem is confused by the presence of injuries to other body cavities and/or the extremities. Often, too, there has been injury to several organs. While the need for an emergency abdominal exploration should not be an issue following penetrating injuries, the same is not true following nonpenetrating (or closed) injuries. The nonpenetrating injuries usually bring up the critical question: "Should this patient be explored?" While it is true that abdominal exploration should never be regarded as a harmless procedure, it is also true that, when indicated, operation constitutes a diagnostic and resuscitative measure for which there is no substitute.

Nonpenetrating injuries of the abdomen are due to direct or indirect violence, usually in the form of crushing, tearing or compressing trauma. Both the solid and hollow organs of the abdominal cavity may be damaged. Other factors being equal, the degree of visceral injury will depend on the mobility or fixation of specific organs within the abdomen, their friability and vascularity, their compressibility or distensibility, their soundness (e.g., a stomach which is ulcerated or a spleen which is enlarged is more likely to be ruptured than the normal organ), the protection afforded by the covering structures and the relative vulnerability of the blood supply to damage. To some extent, also, the duration with which the force acts determines the severity of the visceral injury Sudden, severe compressing forces are more likely to produce extensive internal injury than longer-acting stresses of similar magnitude. The hollow organs are most likely to be ruptured when distended. Tears of the duodenum, ileocecal region or mesentery can occur from closed injuries because these structures are

his opinion as the patient's clinical course requires, and he will request consultation as often as necessary. He will also withhold morphine or other drugs which may confuse the clinical picture until a decision relative to definitive care can be made. Furthermore, he will appreciate that the early signs after injury are the result of damage to covering structures, such as skin, muscles and parietal peritoneum, while those which appear later result from extravasation of blood or other body fluids, bowel content, etc., which incite peritoneal irritation and peritonitis. During the period of *delusive calm* which often precedes the development of peritonitis, the opportunity for surgical repair is greatest. The risks to the patient are often least when the surgeon is guided by the philosophy that "it is sometimes better to look and see rather than to wait and see."

History

When a patient has received a penetrating injury to the abdomen, all pertinent information relating to the circumstances surrounding the injury and the nature of the object causing the injury should be noted. In gunshot wounds, the position of the person injured and the position of the assailant should, if possible, be determined. In stab wounds, knowledge of the length of the blade or instrument, the force with which it was introduced, the direction in which it was thrust, etc., may be helpful. The immediate and subsequent course of the patient and any first-aid treatment which has been given should also be recorded. Following closed injuries of the abdomen, the details of the traumatic episode should also be documented, including those items which relate to the medicolegal aspects of the case.

Physical Examination

After securing the history and provided the patient's condition permits, a rapid physical examination should be made. Obviously, if the patient's general condition is critical, immediate resuscitative measures are in order and detailed inquiry must be postponed. The physician should carefully observe the following procedure: When there are perforating injuries, observe the wounds of entrance and exit, since the location of these wounds may indicate the extent of injury. Observe the wounds for extravasation of blood or other fluids. Palpate the abdomen for localized pain, tenderness and muscle spasm.

generally a fruitless, and possibly a damaging, procedure. It really tells nothing. All patients with penetrating wounds of the abdomen should be explored. In the case of knife wounds, a limited exploration may be in order.

Blast injuries usually result from the detonation of explosives either in air or in water. They are characterized by diffuse and extensive visceral damage with or without gross damage to the surface tissues. The contents of all body cavities may be disturbed by the sudden and violent compression and decompression waves which follow a blast. The extent of the injury produced is related to the magnitude of the explosive force, the distance the individual is from the explosion and his position at the time, the protection afforded by the environment, and the compressibility of the medium in which the explosion occurs. Explosions under water tend to inflict greater damage to the body than those in air. In most instances, after severe blast injury there will be diffuse combined injuries to the cranial, thoracic and abdominal organs without localization.

Clinical Considerations

There are certain general principles in the management of patients with abdominal injuries which must be emphasized. The condition of the patient as a whole must be assessed without overemphasis on local or obvious injuries. The physician should not allow his attention to be diverted by the presence of a facial laceration, extremity fracture, head injury, chest injury, etc, while less obvious serious injuries to the abdomen are overlooked. Unless the abdominal injuries are recognized and treated early, the patient may be lost from shock, hemorrhage and infection. It is, therefore, necessary to suspect the possibility of intra-abdominal injury and to remain alert to the development of significant signs and symptoms after any acute trauma.

A period of observation may be indicated before the issue can be settled. It is essential that the same observer take the responsibility for checking and evaluating the patient's clinical course during this critical period. The physician must repeatedly look for changes in signs and symptoms which indicate progression, regression or continuance of local and systemic disturbances. He must institute necessary diagnostic and therapeutic measures, realizing, all the while, that the natural response and the response to treatment may re-establish the steady state and further cloud the issue. He must reserve the right to change

be little change in the red cell count, the hemoglobin value and the hematocrit. A rapid rise in the total leukocyte count and the percentage of polymorphonuclear cells follows most traumatic injuries. Serial determinations of the blood values at three or four hour intervals are helpful during observation and can often be correlated with the patient's clinical course.

TREATMENT

Treatment of shock is of first importance. Plasma expanders should be given until whole blood is ready. It should be made sure that sufficient blood is available to support the patient throughout the critical phases of treatment. Four or more units of blood (2,000 ml.) are often required for the patient in severe shock before operation can be undertaken. If the patient fails to respond to rapid transfusions of blood, the physician should suspect continued blood loss and consider the need for emergency operative intervention. Oxygen administration by a nasal catheter and sedation may also be indicated. Morphine must be used cautiously, if at all. The patient should receive antibiotic drugs and antitetanus serum or toxoid in amounts previously detailed.

If the patient has a penetrating injury of the abdomen and the response to resuscitative measures is unsatisfactory, operation should be undertaken immediately. If the patient responds to these measures, operation should be undertaken as soon as the general condition has stabilized. If the patient has a nonpenetrating injury of the abdomen and there is reasonable evidence to indicate visceral damage, operation should be undertaken as soon as the patient's condition permits. If, on the other hand, the indications for exploration are not clear, a short period of observation, usually not to exceed six hours, may be advisable. At the end of this time, a definite decision regarding operation should be made. The relationship between the length of time from abdominal injury to operation and the postoperative morbidity and mortality is a linear one.

The signs which suggest the presence of visceral injury and the need for exploration are:

1. Localized abdominal pain, tenderness, and muscular rigidity which persist, increase or become diffuse
2. Absence of peristalsis, as indicated by absence of bowel sounds
3. Evidence of shock
4. Persistent vomiting
5. Blood in the vomitus, stool or urine

Note fulness, masses and distention, which can result from extravasation. Observe the patient carefully for signs of blood loss, such as hypotension, tachycardia, pallor or the signs of established shock. Auscultate the abdomen for signs of bowel activity, and in the absence of bowel sounds suspect intraperitoneal injury. Note the degree to which respiratory movements of the abdomen are impaired, and always consider the possibility of combined abdominothoracic injury. All of these changes should be recorded, checked and rechecked frequently during a period of observation, if necessary.

If the patient has vomited blood or has passed blood in the urine or feces, the suspicion of serious intra-abdominal damage is heightened. In any case, pass a nasogastric tube and empty the stomach to determine if blood is present. The urine must also be examined for blood; and if the patient is unable to void, he must be catheterized. With rupture of the urethra, the catheter usually will not enter the bladder. With rupture of the bladder, no urine, or only a small amount of bloody urine, will be obtained. If bladder rupture is suspected, a measured amount of sterile isotonic saline solution (about 250 ml.) is introduced, if it cannot be recovered, rupture of the bladder is likely. Cystograms are then in order. Blood in the stool or demonstrated on rectal or sigmoidoscopic examination suggests an injury to the bowel.

X-ray Examination

The x-ray examination of the patient who has suffered abdominal injury usually consists of a flat and upright abdominal film and a chest film. Free air in the peritoneal cavity is almost pathognomonic of rupture of a hollow organ. Localized areas of increased density (extravasation), displacement of hollow organs, foreign bodies (e.g., bullets or shell fragments), obliteration of the psoas shadow or herniation of the diaphragm are all significant findings. Special x-ray studies include intravenous pyelography and cystography. In general, x-ray studies with barium should not be made. The physician should be cautious about putting too much faith in a single set of negative x-rays. The x-ray examination is only one aspect of the clinical evaluation, and, as with other observations, time brings changes.

Laboratory Studies

Laboratory studies, with the exception of those of the blood and urine, are usually of little help. Until hemodilution occurs, there may

be little change in the red cell count, the hemoglobin value and the hematocrit. A rapid rise in the total leukocyte count and the percentage of polymorphonuclear cells follows most traumatic injuries. Serial *determinations of the blood values at three or four hour intervals* are helpful during observation and can often be correlated with the patient's clinical course.

Treatment

Treatment of shock is of first importance. Plasma expanders *should be given until whole blood is ready. It should be made sure* that sufficient blood is available to support the patient throughout the critical phases of treatment. Four or more units of blood (2,000 ml.) are often required for the patient in severe shock before operation can be undertaken. If the patient fails to respond to rapid transfusions of blood, the physician should suspect continued blood loss and consider the need for emergency operative intervention. Oxygen administration by a nasal catheter and sedation may also be indicated. Morphine must be used cautiously, if at all. The patient should receive antibiotic drugs and antitetanus serum or toxoid in amounts previously detailed.

If the patient has a penetrating injury of the abdomen and the response to resuscitative measures is unsatisfactory, operation should be undertaken immediately. If the patient responds to these measures, operation should be undertaken as soon as the general condition has stabilized. If the patient has a nonpenetrating injury of the abdomen and there is reasonable evidence to indicate visceral damage, operation should be undertaken as soon as the patient's condition permits. If, on the other hand, the indications for exploration are not clear, a short period of observation, usually not to exceed six hours, may be advisable At the end of this time, a definite decision regarding operation should be made. The relationship between the length of time from abdominal injury to operation and the postoperative morbidity and mortality is a linear one.

The signs which suggest the presence of visceral injury and the need for exploration are:

1. Localized abdominal pain, tenderness, and muscular rigidity which persist, increase or become diffuse
2. Absence of peristalsis, as indicated by absence of bowel sounds
3. Evidence of shock
4. Persistent vomiting
5. Blood in the vomitus, stool or urine

6. Free air in the peritoneal cavity
7. Progressively increasing leukocytosis
8. Continued hemorrhage, as indicated by the blood findings and poor response to transfusions

Operative Care

If operation is required, it should be done as soon after injury as the patient's condition permits. All penetrating wounds should be débrided, irrigated and closed or left open, according to the local wound findings. A separate abdominal incision should be made for exploration. The location and type of the incision will depend on the nature of the injury and the preference of the operator. It is essential that a complete exploration of the abdomen be carried out when even a remote possibility of multivisceral damage exists. The exploration must be systematic and all-inclusive, and can properly be done only under direct vision. Damage to retroperitoneal organs is frequently overlooked. In order to avoid the errors of omission after gunshot wounds, for example, inspection should be made of all portions of the gastrointestinal tract, the liver, gallbladder and bile ducts, the pancreas and spleen, the kidneys, ureters and bladder, and the mesentery and omentum. Usually the perforations that are missed at operation will be discovered at autopsy.

The simplest methods, which insure closure of perforations while maintaining continuity of the gut, should be used. Wounds of the unprepared colon are treated by exteriorization, closure and proximal colostomy, or by primary closure, according to the circumstances. Areas of bowel which have been devitalized as a result of injury to the mesentery must be removed. Perforations of the bladder must be closed and bladder drainage established. Extensive lacerations of the liver must be debrided, bleeding must be controlled and drainage established to provide for the escape of bile. Lacerations of the spleen require splenectomy. Wounds of the gallbladder or bile ducts require closure and drainage. Lacerations of the kidney require débridement, control of bleeding and drainage. Immediate nephrectomy is rarely necessary All free and clotted blood, detached portions of viscera, and gastrointestinal fluid collections should be removed from the peritoneal cavity. Foreign bodies should be removed insofar as possible. Intraperitoneal drainage is rarely necessary. Antibiotics are generally not introduced into the peritoneal cavity.

Postoperative Care

The many items advocated for the care of patients after elective abdominal operations are applicable to those who have sustained abdominal injury. Fluid, electrolyte and blood-volume levels must be maintained. Nutritional intake is usually deficient during the early recovery period; but with resumption of oral feeding, nutritional balance is soon restored. The antibiotic agents singly or in combination have materially reduced the mortality of this period. Penicillin and streptomycin have proved highly effective. The broad-spectrum antibiotics also have a definite place in therapy. Of primary importance in the recovery period is the institution of physiologic rest of the gastrointestinal tract. Nasogastric suction (in some instances using the long intestinal tube) is mandatory in the early postoperative period. The intake by mouth should be restricted until bowel activity and bowel continuity are assured. Early ambulation is applicable provided that it is prescribed with discretion. Analgesic drugs and sedatives in small amounts, as required, are needed but should be withdrawn as soon as pain and restlessness are minimal.

Postoperative complications may develop and are related to the nature and extent of the injury. Atelectasis and pneumonia, intra-abdominal abscesses, intestinal obstruction, venous thrombosis and pulmonary embolism can be anticipated in some cases. The physician must be on the alert for their development and use all measures necessary to avert or treat these conditions.

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struggle and cry, thus making evaluation of the abdominal findings difficult or misleading. Another factor which contributes to errors of interpretation relates to the fact that the pain reaction in the infant is poorly developed; also, that in the presence of severe intra-abdominal disease, pain may not be apparent. The total white cell and differential white cell counts are helpful, but not always reliable, indications of infection in very young patients.

Acute Appendicitis

As in adults, acute appendicitis is the most common acute surgical condition of the abdomen in the young. The progression of local pathologic changes may be very rapid and the systemic response severe.

The clinical features of appendicitis in the young are the same as in the adult, *conditioned somewhat* by the unique problems of infancy and childhood. A reliable chronologic history is often unattainable. An illness which begins with a chill, nausea or vomiting, rather than with abdominal pain, suggests a diagnosis other than acute appendicitis. The onset of acute appendicitis may be preceded by an acute upper respiratory infection or other febrile disease; but more often, the child has been well. Abdominal pain, nausea and vomiting and slight constipation are usual.

Localized tenderness over the right lower quadrant of the abdomen is the most important single sign of appendicitis in the infant, child and adult. There is muscle spasm, rebound tenderness, sometimes a palpable mass and less often rectal tenderness and psoas spasm.

When the appendix is suspect and nonsurgical conditions have been excluded from the diagnosis, operation is indicated without delay. The common diseases of the young to be differentiated from acute appendicitis include: acute gastroenteritis; acute mesenteric adenitis; acute pyelonephritis, acute infectious exanthemas, especially measles, upper respiratory tract infections; right lower lobe pneumonia, and diabetic acidosis.

Appendectomy should be performed, if possible, as soon as the diagnosis has been made and fluid and electrolyte balance has been restored. Drainage of an established appendical abscess and interval appendectomy (six weeks to two months after the acute illness) is sometimes advisable in advanced cases. Primary conservative or nonoperative treatment is rarely indicated. The muscle-splitting

CHAPTER 24

Abdominal Conditions of Infants and Children

The child is never too sick to be despaired of, nor too well to be sure of.—HUGHES.

ONE MUST bear in mind that the infant or child cannot be treated as a small man or woman. The progression of disease processes and the systemic responses are always exaggerated in the very young. The rate at which pathologic changes occur may be accelerated by misguided parental interference. It was in regard to this that Moynihan referred to the "therapeutic peritonitis" which often complicates appendicitis after a cathartic has been given. The delicately balanced homeostatic mechanisms of the infant are easily upset. Severe degrees of dehydration, electrolyte depletion and acid-base imbalance rapidly develop with failure of intake and/or unusual fluid loss. A favorable factor influencing recovery is the remarkable recuperative power of the infant and child when given proper treatment.

Early and correct diagnosis may be very difficult. The infant or young child is unable to describe symptoms or to give a reliable history, and the parent frequently can offer no more than a fragmentary story of the illness. Thus, physical diagnosis in pediatric cases is of the first importance. When signs and symptoms suggest an acute intra-abdominal surgical condition, it is particularly important to examine carefully and to rule out disturbances involving other areas, such as those of the skin, throat and ears, chest and urinary tract, which sometimes simulate abdominal disease. It should be remembered that often fear and uncertainty will cause the child to

cated by profound systemic disturbances and local gangrene of the bowel, treatment is difficult.

The story which the mother gives regarding the infant is usually quite typical. He awakens with a cry and soon has recurrent episodes of severe abdominal pain which cause him to double up and to scream for five or ten seconds or more. He may sleep and appear to be normal between attacks. Early in the course of the disease there is vomiting, which soon becomes persistent. A small "currant jelly" stool,

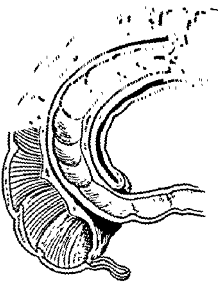

FIG 105.—Ileocolic intussusception. The terminal ileum has passed through the ileocecal valve into the cecum and ascending colon. Intestinal obstruction and strangulation of the ileal blood supply occur.

composed of mucus and blood, may be passed. Severe abdominal distention develops, and signs of shock appear as the condition progresses.

A sausage-shaped mass is often palpable in the upper right quadrant and is almost pathognomonic for intussusception. The right lower quadrant may also feel "empty." If the intussusception has passed into the distal part of the colon, a mass which resembles the cervix of the uterus may be palpable in the rectum.

The x-ray films of the abdomen reveal evidence of small-bowel distention with fluid levels. It should be remembered that scattered gas in the small intestine is a normal finding in infants. A filling defect in the cecum or colon and failure of the barium to enter the small bowel can be demonstrated by barium enema.

McBurney incision is used almost routinely. Systemic antibiotic therapy is essential if there has been peritoneal soiling.

Acute Mesenteric Adenitis

Mesenteric lymphadenitis may be indistinguishable from acute appendicitis until the time of operation. There is mild to moderate, diffuse or localized, right lower quadrant pain, often with little or no nausea or vomiting. Abdominal tenderness is usual, but muscle spasm is often absent. There may be a leukocytosis with a relative increase in the mononuclear cells. Fever and leukocytosis may be out of proportion to the abdominal findings.

Operation clarifies the diagnosis, and appendectomy sometimes appears to accelerate recovery. The cause of acute mesenteric lymphadenitis has not been established. The operative findings are: (1) no definite pathologic changes in the appendix; (2) large, soft lymph nodes in the ileocecal mesentery, which show no growth on culture; and (3) usually a small amount of free peritoneal fluid which is sterile on culture.

Acute Intussusception

Intussusception of infancy and childhood occurs predominantly in the first two years of life and in boy babies. The cause is obscure. An associated mesenteric adenitis is frequently encountered at operation. It is probable that some disturbance in intestinal motility initiates intussusception.

The intussusception usually begins at the ileocecal sphincter (Fig. 105): the ileum inverts into the cecum and progresses into the colon—graphically described as "the big bowel eats the small bowel." The abnormal bolus (intussusceptum) may traverse the entire length of the large bowel (intussuscipiens) and be palpable in the rectum, or may even protrude from the anus. A Meckel's diverticulum is occasionally responsible for initiating intussusception. In adults a pedunculated polyp or a carcinoma, usually located in the sigmoid colon or small intestine, is almost invariably the underlying initiating lesion.

The clinical picture is that of acute small-bowel obstruction. The duration of obstruction before treatment is the most important factor influencing prognosis. In intussusception of short duration, the treatment is relatively simple; but when it is of long standing and compli-

cated by profound systemic disturbances and local gangrene of the bowel, treatment is difficult.

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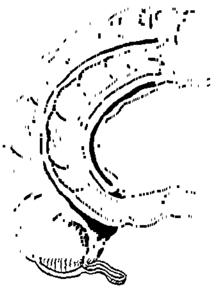

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The treatment of acute intussusception is as follows:

In the early case and under careful fluoroscopic control, reduction of the intussusception can often be accomplished by means of a barium enema. The hydrostatic pressure should be carefully controlled and should not exceed that obtained by elevating the enema can more than 3 feet above the patient. Particular attention should be given to complete reduction of the intussusception as demonstrated by barium "flooding" into the small bowel and disappearance of the palpable abdominal mass.

If reduction by hydrostatic means is considered hazardous because the possibility of strangulation of bowel exists, or if the reduction is incomplete or inconclusive, operative treatment is in order.

After preoperative preparation, including a venous cut-down, fluid-electrolyte and blood-volume replacement, the operation is undertaken. The intussusception is reduced by uniform pressure on the distal part of the mass, rather than by traction on the small bowel. Thus the small bowel is expressed from the colon by compression. The chances of recurrence of intussusception are slight, and it is usually unnecessary to anchor the small bowel by suture. The appendix is not removed routinely, but it may be removed if this does not appear to add to the surgical risk. If the bowel has been irreparably damaged by strangulation of its circulation, it must be resected.

Congenital Hypertrophic Pyloric Stenosis

This condition is most often seen in male infants from three to ten weeks of age. The onset of symptoms commonly occurs about the sixth week. There is pylorospasm with hypertrophy of the musculature of the pyloric sphincter The cause is unknown.

The cardinal symptoms and signs of hypertrophic pyloric stenosis are: projectile vomiting, left to right visible peristalsis, a palpable abdominal tumor (olive shaped), failure to gain weight and obstipation.

Conditions which must be differentiated from plyoric stenosis are: duodenal atresia or stenosis, congenital atresia or midgut volvulus, Meckel's diverticulitis with obstruction, and functional pylorospasm. It is necessary to distinguish between pyloric obstruction and intestinal obstruction. The vomitus contains bile if the obstruction lies beyond the papilla of Vater, while bile is unusual in obstruction at the pylorus. Radiographic study of the upper gastrointestinal tract may be indi-

cated but is not always necessary. *It is sometimes difficult to differentiate between pyloric stenosis and pylorospasm.*

In treating congenital pyloric stenosis, the fluid and electrolyte balance *should first be restored. Small whole-blood transfusions may* be indicated. Fredet-Rammstedt pyloromyotomy should then be performed. In this operation, an incision is made longitudinally through the serosa of the pyloric tumor, and the hypertrophied muscle fibers are separated down to the submucosa with a small hemostat until the mucosa and submucosa bulge outward through the opening in the muscle. Thus an open pyloric canal is established. It is important to break all of the muscle fibers but to avoid opening the duodenum. The defect in the muscular "olive" heals by fibrosis, and later the hypertrophied muscle atrophies. The results are excellent.

Meckel's Diverticulitis

A Meckel's diverticulum is a remnant of the embryonic vitelline duct. It is an uncommon anomaly, present in only about one in a hundred individuals. It may be the site of inflammation, obstruction or *ulceration. Ulceration is usually the result of erosion caused by acid* peptic secretion elaborated by ectopic gastric glands located in the mucosa of the diverticulum.

The symptoms of acute Meckel's diverticulitis sometimes resemble those of acute appendicitis. Patients subjected to appendectomy should always be examined for a persistent Meckel's diverticulum unless the appendix has ruptured or the condition of the patient precludes further exploration. If present, the diverticulum will be found in the ileum within 24 inches of the ileocecal sphincter.

Recurrent severe hemorrhage from the intestinal tract in infants or children may result from Meckel's diverticulitis. Roentgenologic studies are generally of little help in establishing the diagnosis. If bleeding is severe or continued, exploration should be undertaken after whole-blood transfusions *have been given. For correction* of the condition, the diverticulum should be excised and the opening closed in such a manner that the lumen of the bowel is not narrowed.

Duodenal Atresia and Stenosis

Segments of the intestine may fail to develop a lumen. If the duodenum is atretic, signs of a high intestinal obstruction appear as

soon as feedings are begun. According to the level of the obstruction, the vomitus will or will not contain bile. There will be jaundice if the ampulla of Vater is involved. X-ray studies are an aid in diagnosis. Early operative treatment is essential.

Congenital Atresia of the Bile Ducts

Incomplete canalization of the extrahepatic bile ducts will result in persistent and progressively increasing jaundice with acholic stools. If there is atresia of the common bile duct, the gallbladder will be distended and a cholecystogastrostomy or cholecystoenterostomy is required for diversion of bile to the intestine. Surgical relief of the obstruction is very difficult, or impossible, if there is atresia of the common hepatic duct or the intrahepatic ducts.

Imperforate Anus

Imperforate anus is due to a failure of the proctodeum (anal membrane) to undergo spontaneous rupture or to failure of development of the hindgut. As a consequence, there is no communication between the rectum and the anal canal. There may be a thin anal membrane or a total absence of a canal between the perineum and the rectum. Anorectal anomalies are easily recognized at birth (Fig. 106). Imperforate anus is often encountered in association with other congenital anomalies—e.g., rectovaginal fistula (female), rectovesical fistula (male), cardiac malformations, etc. If the rectal closure is complete, there is rapidly developing intestinal obstruction.

Early surgical intervention is required when a complete block exists (Fig. 107). The distance between the anal dimple and the lower rectum can be determined by x-ray studies with the infant in the upside-down position. An opaque marker is applied with slight pressure to the anal dimple. Gas in the rectum will rise to the highest point, and the distance between the external skin marker and the gas bubble in the rectum will indicate the approximate extent of the atretic area.

If the membrane is very thin, it can be opened under light anesthesia. First, a needle is passed through the membrane and guided posteriorly (not anteriorly) toward the coccyx and sacrum. When gas has been aspirated from the bowel, the opening can be enlarged, but great care must be taken to prevent injury to the anal sphincter mech-

anism whi[illegible]

When [illegible] ing fistulas, the problem is more serious and special surgical treatment is required. An abdominoperineal operation for the establishment of

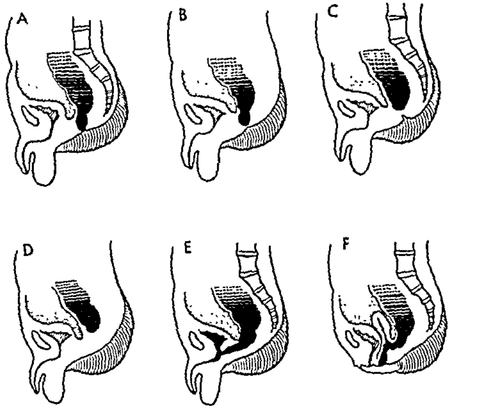

Fig. 106.—Anorectal anomalies of the newborn. *A*, stenosis of the anal canal. *B*, imperforate anus with persistence of the anal membrane. *C*, imperforate anus with high-lying rectal pouch; anal depression present. *D*, imperforate anus with high-lying pouch; no anal depression. *E*, imperforate anus with rectovesical fistula in the male. *F*, imperforate anus with rectovaginal fistula in the female.

anorectal continuity may be indicated. This may be done as the initial procedure in some instances. In others, colostomy may be necessary and the definitive operation deferred until conditions are more favorable.

Congenital Megacolon

Congenital megacolon (Hirschsprung's disease) is chiefly a condition of infants and children. It is characterized by severe and obstinate constipation which often results in profound systemic disturbances

due to chronic intestinal obstruction. There is moderate to enormous dilatation and hypertrophy of the colon above a spastic or contracted area which is usually located in the rectum or rectosigmoid region. The narrowed or spastic segment is deficient or devoid of autonomic

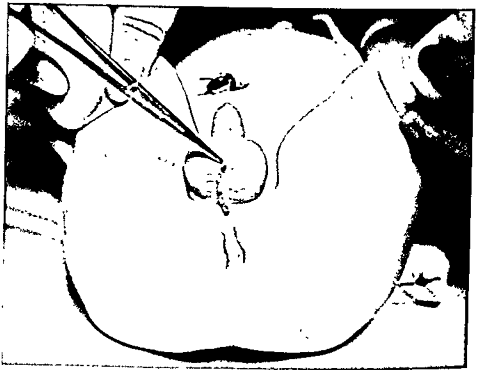

FIG 107.—Imperforate anus in a newborn male infant. A superficial anoscrotal fistula is indicated by the hemostat. Note also the median band of skin across the anus. After it was determined by x-ray studies that the rectal pouch and the anal dimple were separated only by a thin membrane, the membrane and the fistula were excised and the rectal pouch was sutured to the external skin. Recovery was uncomplicated

ganglion cells in Auerbach's plexus, and this lack is believed to be responsible for disturbances in motility. Normal propulsive activity is absent, and a functional obstruction exists. The changes in the colon above the area of functional obstruction (hypertrophy and dilatation) result from an increased work load imposed by the chronic obstruction. The response is similar to that seen with obstruction of any hollow muscular structure. There appears to be no disease of the proximal bowel, which returns to normal size and thickness once the obstruction is relieved.

Most children with megacolon will respond to medical treatment Intractable constipation, chronic distention and malnutrition indicate

the need for surgical treatment (Fig. 108). In the past, numerous operations, including sympathectomy, colectomy, colostomy, etc., were utilized with little success. Today an operation directed at removal of the aganglionic (spastic) area and a portion of the proximal hyper-

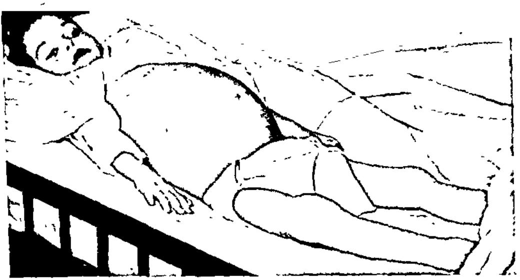

Fig. 108.—Congenital megacolon (Hirschsprung's disease) in a 5 year old child. There was obstipation, abdominal distention and failure to gain, as well as recurrent bouts of acute intestinal obstruction due to fecal impaction. A narrowed spastic area in the sigmoid colon was the site of functional obstruction. The lower colon and rectum were resected, and the bowel was reunited just above the anal sphinter area. The sigmoid colon was found on histologic examination to be deficient in ganglion cells in the myenteric plexus. The long-term result from the operation was satisfactory.

trophied dilated bowel and low anastomosis of the colon to the anus is widely used (Swenson's operation). The follow-up results have been quite satisfactory.

Malrotation of the Midgut with Midgut Volvulus

The process by which the midgut segments are formed and fixed in their normal position is very complex and difficult to visualize. It occurs by a progression of three-dimensional changes during the fifth to twelfth weeks of embryonic life. With but minor exceptions, the anatomic relationships are established during this period. When complete the duodenum then lies below the superior mesenteric artery; the intestinovitelline duct is disappearing; the cecum, appendix and as-

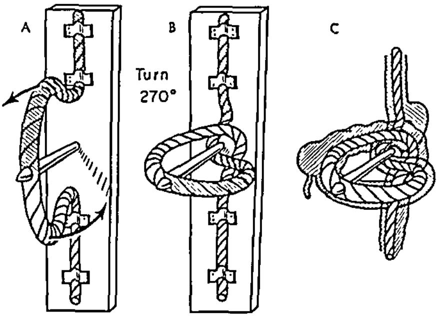

FIG. 109.—Normal rotation of the midgut. This phase of development can be illustrated by a simple model, as suggested by Snyder and Chaffin. The model is

jejunum and the proximal ileum are formed. The rod corresponds to the superior mesenteric artery, which is the axis of rotation. The lower, unshaded, portion of the loop corresponds to the segment of the midgut from which the terminal ileum, the cecum and appendix, and the right colon are formed. The knob opposite the rod corresponds to the intestinovitelline duct. *B*, the loop has been rotated counterclockwise through an arc of 270 degrees. The top portion of the loop has now become the bottom one, and the bottom one the top. Note that the shaded segment passes beneath the rod and the unshaded segment passes over the rod. *C*, the divisions of the alimentary canal have been sketched in relation to the rope The duodenum pa
Meckel's d
drawing),
(From Snyder, W. H., Jr., and Chaffin, L.: Embryology and pathology of the intestinal tract: presentation of 40 cases of malrotation, Ann. Surg. 140:368-379, September, 1954.)

cending colon lie in the right side of the abdomen; and the transverse colon lies in front of the duodenum.

The following may help to explain how rotation comes about. The primitive alimentary canal is a cordlike structure which later acquires a lumen. It is suspended by a dorsal mesentery carrying the blood supply. The midgut receives its blood supply from the superior mesenteric artery, which represents the axis of rotation later on. During the early period of rapid growth, the midgut is crowded out of the abdominal cavity and into the cord, producing a physiologic herniation. Shortly thereafter, orderly reduction of the bowel back into the abdominal cavity takes place, the last portions to leave being the first portions to return. As reduction of herniated bowel occurs, a process of rotation is observed (Fig. 109) which provides for the systematic distribution and eventual fixation of the several parts of the midgut. The essential elements of rotation concern: position of the superior mesenteric artery, which represents the axis of rotation and which extends from the posterior abdominal wall, anteriorly; turning of the midgut, counterclockwise, through an arc of 270 degrees (three quarters of a circle); and lastly, peritoneal fixation of the duodenum, the small bowel mesentery, the cecum and the ascending colon.

When rotation is complete, several portions of the midgut become fused to the posterior abdominal wall; other portions remain attached by mesenteries. With failure of rotation, abnormal lines of peritoneal fusion and lack of anchorage of the small-bowel mesentery occurs. The common findings in midgut volvulus are: obstruction to the duodenum due to adhesions, and twisting (volvulus) of the small bowel clockwise on its narrow-based mesentery. Other conditions resulting from errors in midgut development are: omphalocele (persistence of the embryonic hernia), Meckel's diverticulum and various types of atresias of the gut.

The clinical findings in malrotation of the gut are those of intestinal obstruction. Signs of obstruction usually appear shortly after birth, but they may not develop until later in life. In newborn infants with obstruction, the physician should look for imperforate anus and atresia of the rectum and colon. Also, he should consider ileocolic intussusception in all infants and children. X-ray films of the abdomen may suggest the site of obstruction. A barium enema is in order if large-bowel obstruction is a diagnostic possibility.

Malrotation with obstruction is an emergency condition, and surgical treatment is imperative. Before operation, attention must be

given to the systemic needs of the infant, but little delay is permissible. With volvulus of the midgut there is progressive necrosis of the bowel, and the infant's chances for survival fall off sharply when this complication occurs.

The operation consists of unwinding the twisted bowel, as well as freeing the adhesions which block the duodenum. No attempt should be made to suture the segments of the midgut in their normal positions. Necrotic bowel, if present, must be removed, and continuity of the alimentary canal restored by anastomosis.

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CHAPTER 25

The Head and Neck

MOUTH, TONGUE, JAWS AND SALIVARY GLANDS

CONGENITAL DEFECTS

THE COMPLEX developmental processes which result in the formation of the mouth and jaws often lead to congenital abnormalities or defects. The most common of these imperfections are those of the upper lip and the palate, resulting in cleft lip (harelip) or cleft palate, or both. The surgical repair of a cleft lip should be undertaken early, usually in the first few weeks of life; but the repair of a cleft palate may be deferred until the second or third year of age, before serious speech difficulties have arisen. Proper surgical management results in excellent anatomic and functional results.

TRAUMATIC INJURIES

The management of injuries to the lips, tongue and jaw is essentially similar to that in other regions. Wounds of these structures are attended by moderate to severe hemorrhage and by some degree of infection, but usually good healing occurs. While contamination with mouth organisms does occur, serious infection is, fortunately, unusual. Closure of wounds in this area requires more than the usual attention to cosmetic aspects. Proper initial handling of the wound will often obviate the need for secondary plastic procedures. Injuries which are associated with division of the branches of the facial nerve, laceration of a salivary gland, penetration into the mouth, fracture of the jaw or serious laceration of the tongue require specialized surgical care. The timing of operation, selection of anesthesia, positioning of the patient, maintenance of the airway (tracheotomy may be necessary) and re-

duction of fractures are problems of immediate concern. Immediate repair should be carried out only if the patient's general condition is satisfactory.

Infections

Infections in this area are commonly saprophytic and noninvasive in behavior. Invasive infection may follow acute dental infections, acute tonsillitis and pharyngitis. Suppuration of the parotid gland occasionally occurs as a complication after abdominal operations. Rarely is acute parotitis encountered as a complication of duct obstruction from a salivary stone. Infections of the upper lip and the nose (dangerous areas of the face) may extend through the angular veins to cause cavernous sinus thrombosis. For this reason, infections of the "dangerous areas" should be treated conservatively and not incised, squeezed or otherwise manipulated during the acute phase. The same may be said about all acute infections of the face, the oral cavity, the teeth and the jaws. The extraction of a tooth during the acute phase of dental infection may lead to osteomyelitis or other complications. Osteomyelitis of the jaw may indicate the need for conservative drainage of the soft tissues and later removal of a sequestrum, rather than primary radical treatment.

Benign Tumors and Cysts

A wide variety of pathologic entities is found in the mouth and jaws, but only a few of the more common conditions can be described here.

Ranula.—The ranula ("little frog") is a soft, painless, semitransparent swelling beneath the mucous membrane of the mouth. It usually appears at the side of the tongue and may extend back to the angle of the jaw. The cyst, which contains a ropy mucoid fluid, arises from submucous glands and sublingual ducts. Excision is the treatment of choice; but if complete removal is not possible, packing with irritant material or marsupialization may lead to obliteration.

Dermoid Cyst.—This cyst is rather firm and usually contains hair, sebaceous material and debris. It occurs in the midline of the floor of the mouth and should be differentiated from the ranula, which develops to the side of the tongue.

Epulis.—The epulis is a tumefaction of the dental margin rather

than a pathologic entity. It may develop from the dental periosteum and appear from the root membrane attached to the alveolar process. It may occur in regions where teeth have recently been lost or extracted. The tumor is soft, red and friable, or firm and pale, according to whether vascular or fibrous tissue predominates. Three clinical types are described: fibroma, fibroangioma and giant cell epulis. Treatment

FIG. 110.—Carcinoma of the parotid gland. The patient had been aware of a marble-like mass in the parotid for many years. In recent months it had enlarged rapidly and produced paralysis of the seventh cranial nerve. Radical local excision was performed, but the tumor recurred about a year later. Radiation therapy was instituted with little effect. It is probable that the cancer arose in a "mixed tumor."

consists of excision of the tumor, with extraction of a tooth on either side of the lesion and thorough curettement of the tooth socket and bone.

ODONTOMA.—An odontoma is a bizarre mass of aberrant tooth forms enclosed in a fibrous capsule or cementum. Odontomas vary in size and shape and are usually buried in bone. They do not require removal unless they produce symptoms. Two types are encountered—the hard and soft varieties. The hard type is probably a malformation. The soft type is a true neoplasm of mesodermal origin.

DENTIGEROUS CYST.—Dentigerous cysts probably grow from a

dental follicle of an unerupted tooth. They usually contain a rudimentary tooth composed of the basic tooth elements: dentine, cementum and enamel. These cysts are painless and may attain considerable size. Dental root cysts are the result of infection following the death of the pulp of the erupted tooth. The cyst may remain after the extraction of the tooth.

ADAMANTINOMA.—The adamantinoma, or ameloblastoma, is derived from the enamel organ and usually appears in the molar region. The growths are large, bulky, lobulated and honeycombed. They are benign but locally infiltrative and destructive. The roentgenologic appearance may be diagnostic. The condition is treated by wide excision, including a portion of the jaw. Unless completely removed, this tumor will always recur.

MIXED TUMORS.—These tumors occur chiefly in the parotid gland (Fig. 110) and less often in other salivary glands or in the mucous membrane of the palate or lip. They are slow-growing, hard, often nodular and painless. Characteristically, they are located over the parotid or behind the angle of the jaw. They may contain epithelial elements, fibrous tissue, cartilage, bone, lymphoid tissue, fat and striated muscle. They are generally benign but often exhibit malignant characteristics after repeated but incomplete attempts at local removal. Recurrence is common because simple enucleation of the mass is performed and remnants of the tumor are allowed to remain. The potential seriousness of mixed tumors and the need for more radical treatment is not commonly appreciated. In order to cure this condition, it is usually necessary to remove the entire superficial lobe of the parotid or, in some instances, the entire parotid. Special precautions *must be taken during the dissection to protect the facial nerve from* injury. Only when the tumor is malignant is it justifiable to sacrifice the nerve.

MALIGNANT TUMORS

Primary carcinomas of the lower lip, tongue and oral mucous membrane are relatively common. They are predominately squamous cell (epidermoid) in type and appear to have a common etiologic basis. Chronic irritation resulting from, or in association with, dental snags, carious teeth, poor oral hygiene, ill-fitting dentures, tobacco and syphilis frequently leads to leukoplakia, papillomas, fissures or ulcers, which often appear to be the starting point for cancer. Leukoplakia

is a precancerous lesion which appears as white patches resembling white enamel paint on mucous membranes. It is essential that the physician recognize and correct sources of chronic irritation and potential malignant growth.

Cancer of the lip and mouth spreads directly to bone and muscle and indirectly to distant areas through the lymphatics. Often the local process can be cured or controlled, but metastatic spread results in the death of the patient. Even in the early and apparently favorable cases, it is important to recognize that lymphatic spread may already have

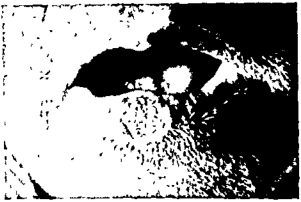

FIG. 111.—Epidermoid carcinoma of the lower lip. Note the location at the vermilion border, the rolled edges at the margin and the central ulceration. Cured by V excision.

occurred and to give careful consideration to the distant areas as well as the local lesion. Too often, attention is focused entirely on the primary tumor. As in other regions, the opportunity for cure of cancer is greatest at the time of initial treatment.

The lymphatic channels near the midline of the lower lip, tongue and oral cavity are crossed. The suprahyoid nodes (submental and submaxillary groups) drain to the upper deep cervical nodes, which lie close to the bifurcation of the common carotid artery.

Primary spread of carcinoma of the upper and lower lip, the anterior portion of the tongue, the alveolar ridges and the buccal mucosa occurs to the suprahyoid nodes. Secondary spread occurs to the upper deep cervical nodes from the suprahyoid nodes, or via collateral channels or retrograde flow.

Primary spread to the deep cervical nodes occurs from the posterior portions of the tongue, floor of the mouth, tonsil, palate, lower pharynx and larynx.

The absence of enlarged nodes is no assurance that metastatic spread has not taken place. Conversely, enlarged nodes do not necessarily indicate the presence of metastases. Inflammatory changes in lymph nodes often simulate changes produced by metastases. The diagnosis of metastatic spread to the regional nodes is, therefore, always open to question unless it is based on histologic evidence. Certain clinical findings, such as induration, multiple nodal involvement and fixation, may indicate to the experienced observer that spread has actually occurred. Between these two extremes—that is, absence of clinical changes and gross clinical changes—many possibilities for mistakes in judgment and management exist.

Carcinoma of the Lip.—Cancer of the lower lip (Fig. 111) occurs almost exclusively in men. Although the lesion may appear as a wart, a fissure or a nodule, most often it is an ulcer with raised margins and a granulating base. Spread to the regional nodes occurs relatively late in the course of the disease, as compared to cancer of the tongue. The prognosis for cure is good, provided metastatic spread has not occurred.

The first step in the treatment is to establish the diagnosis by biopsy. The local lesion can be completely removed by excision or destroyed by radiotherapy. The surgical treatment consists in a wide V excision with plastic repair of the lip. Bilateral dissection of the lymph nodes of the upper neck (supraomohyoid) may also be indicated. It may be done at this time or deferred until later. When supraomohyoid dissection is done in the absence of palpably enlarged nodes, it is sometimes called "prophylactic" neck dissection. There is evidence to indicate that routine prophylactic neck dissection does not increase the cure rate of cancer of the lip. When regular and frequent follow-up observations of the patient cannot be assured, it is advisable to carry out upper neck dissection in the absence of clinical signs of spread. This would seem to be consistent with the principles of good cancer therapy It is generally recognized that radiation of lymph nodes containing cancer is not curative but only palliative. In some instances, radium therapy is used. Occasionally there will be involvement of the mandible by direct extension from a tumor of the lip or from metastatic cancer in the upper cervical lymph nodes. Under such circumstances, combined jaw resection and neck dissection is sometimes indicated.

Carcinoma of the Tongue.—Cancer of the tongue occurs most frequently in males over fifty years of age. The tumor may appear as

a deep ulcer; a nodular mass which eventually ulcerates; a hard, deep crack; or an extensive infiltration without ulceration. Infiltration of muscle and early spread to the regional nodes occur (Fig. 112). The tumor in the tongue is usually more extensive than indicated by external examination. The location of the tumor influences spread, treatment and prognosis.

Pain may be severe. Weight loss soon appears. Ulceration and hemorrhage occur later. Cancer of the anterior part of the tongue

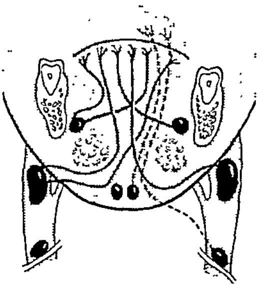

FIG. 112 (*left*).—Carcinoma of the anterior portion of the tongue in an elderly woman Ulceration of the lesion has not yet developed. The patient was treated by irradiation locally and resection of the cervical nodes.

FIG. 113 (*right*).—Lymphatic drainage of the tongue. Note that the channels cross. The lymph node aggregations consist of the submaxillary, submental and upper deep cervical groups (After Rouvière.)

(anterior to the lingual V) offers a more favorable prognosis than cancer of the posterior or infralingual portions. The lymphatic drainage (Fig. 113) from the midline is invariably crossed. The first lymphatic extension from carcinoma of the tongue occurs according to the location of the lesion to the submental, submaxillary or upper deep cervical chain of lymph nodes.

Carcinoma of the tongue must be differentiated from hyperkeratosis, benign papilloma, tuberculosis and primary and tertiary syphilis. Biopsy is essential for diagnosis.

Cancer of the tongue is treated by wide surgical excision (usually hemiglossectomy) or by radiation, either external or interstitial

(radium), according to the location and extent of the lesion. The areas of lymphatic drainage require radical removal because metastatic spread occurs early and regularly in this disease. If the physician waits for unequivocal evidence of nodal involvement, the opportunity for cure is compromised.

CARCINOMA OF THE MOUTH.—Cancer of the floor of the mouth is an unusually unfavorable tumor. Early extension to muscle, bone and lymphatics occurs. The prognosis is similar to that of cancer of the tongue. Treatment consists of radiation to the primary lesion or combined floor of the mouth-jaw resection. Radical neck dissection may be performed simultaneously or after treatment of the local lesion has been completed.

Carcinoma of the alveolar ridge generally involves bone. Involvement of the palate may be primary or secondary to lesions of the paranasal sinuses. Because necrosis of bone often follows intensive radiation of these areas, surgical treatment, with wide excision of the jaw, palate or maxilla, is sometimes advisable.

The most frequent complications of carcinoma of the mouth are distant metastasis, ulceration, hemorrhage, deformity, bone necrosis and osteomyelitis.

The care of the patient with advanced incurable intraoral cancer is difficult even under the best circumstances. Pain is severe, rest is seriously disturbed, food intake is poor, breathing is difficult and sloughing, suppuration and hemorrhage continue until death occurs from exhaustion, pneumonia or exsanguination.

MASSES IN THE NECK

One can do no more than mention some of the features of these tumors which will make one suspicious of what the tumor may prove to be.—LAHEY.

The clinical aspects of masses in the neck constitute an interesting subject for study and speculation. In approaching these problems one must keep in mind the regional anatomy and the fact that the neck contains many tissues and organs (Fig. 114), any one of which may be the site of tumefaction. Here we find skin, fat, fascia, muscle, bone, cartilage, blood vessels, lymphatics, nerves, salivary glands, thyroid, parathyroids, sympathetic nerves and ganglia, carotid bodies and the communicating hollow organs of the respiratory and alimentary tube

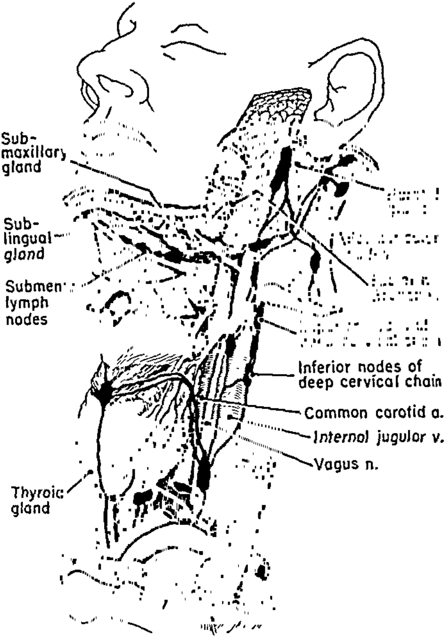

Fig. 114 –Diagram of the deep structures of the neck, as seen from the lateral aspect.

(trachea and esophagus). Each of these tissues and organs may give rise to or be involved in a pathologic process.

The next consideration which would logically follow the regional anatomy would be the regional pathology. Here three broad categories come to mind: malformations, congenital or acquired; infections; and new growths. Congenital malformations are usually present at birth or become manifest in early life. Acquired malformations are often of vascular origin, as, for example, aneurysm or arteriovenous fistula. Infections tend to be acute or chronic according to whether they are due to organisms of the pyogenic or chronic granulomatous type. New growths are of two general types: benign or malignant. Those which are benign originate in and remain confined to the neck. Those which are malignant may originate in the neck and spread to other areas, or originate in other areas and spread to the neck. In certain types of neoplastic disease (e.g., lymphomas), the swelling in the neck may simply be one manifestation of generalized disease.

From the above it is apparent that clinical diagnosis of masses in the neck poses many problems. But much can be learned from analysis of the history and physical findings, together with blood and x-ray studies. The final diagnosis, however, will often depend on biopsy. This is not meant to imply that masses in the neck should be subjected to biopsy immediately in order to obviate the need for other studies. On the contrary, biopsy should be deferred until other studies have been completed, and then performed only when the indications for it are clear.

The most common tumors or cysts of embryologic origin arise from (1) remnants of the median thyroid anlagen (thyroglossal duct) or (2) *remnants of a branchial cleft, usually the second* (branchial cleft cyst). Remnants of both the thyroglossal duct and the branchial cleft may persist as fistulas communicating with the oval cavity and skin or as sinuses opening on the skin.

An understanding of the cervical lymphatics and the regions which they drain is essential to diagnosis. The lymphatics of the neck may be divided anatomically in the superficial and deep systems according to their relationship with the superficial layer of the deep cervical fascia. These systems communicate through many connecting channels. The usual direction of lymph flow is from the superficial to the deep nodes; however, direct and retrograde lymph flow also occur.

The superficial system includes three main groups of nodes: (1) the submental group, which receives lymph from the lower lip and

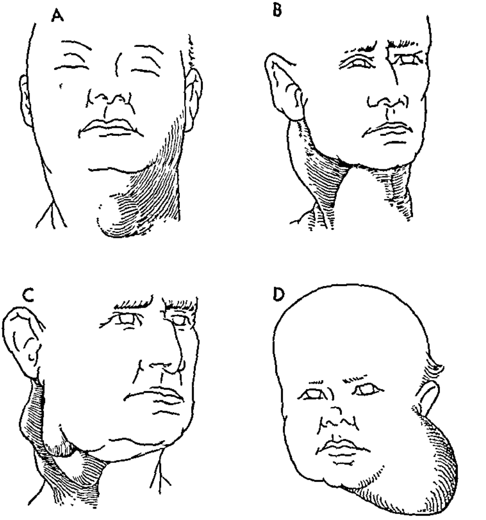

FIG. 115.—Representative masses in the neck—the most commonly encountered types exclusive of thyroid nodules. *A*, thyroglossal duct cyst. *B*, branchial cleft cyst in an adult. *C*, metastatic cancer of cervical nodes from a primary tumor of the floor of the mouth. *D*, cystic hygroma in an infant.

mouth; (2) the submaxillary group, which receives lymph from the floor of the mouth, the upper lip, sinuses and face; and (3) the posterior group (below the angle of the jaw and behind the ear), which receives lymph from the ear and scalp. The superficial system extends downward along the external jugular vein.

The deep cervical system lies beneath the sternocleidomastoid muscle and along the great vessels. The superior nodes of the deep group are located above and around the bifurcation of the carotid artery. They drain the tonsil and the pharynx primarily but are

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consistency, regional changes and associated diseases are the more important points in diagnosis.

THYROGLOSSAL DUCT CYST.—The thyroglossal duct lies in a line between the pyramidal lobe of the thyroid and the foramen caecum linguae. A cyst or sinus tract derived from this structure (Fig. 115, *A*) is located in the midline at any point between the thyroid isthmus and the base of the tongue. The tract is usually attached to, or in close relationship with, the hyoid bone. The cyst may cause no symptoms or may cause symptoms as a result of pressure on other structures. The cyst has a thin wall and is filled with mucoid fluid. Swallowing will cause the mass to move upward. Characteristically, the cyst is movable in a lateral direction but not vertically.

A palpable cord (thyroglossal duct) may be found running upward from the cyst. Because of this duct, the cyst may become infected. If so, the cyst often drains spontaneously, but it may be incised. Ideally, it should be removed before infection occurs. The presence of a thyroglossal duct sinus or fistula complicates treatment. Complete excision of the tract to the base of the tongue, including a portion of the hyoid bone, is necessary for cure.

LATERAL DISCRETE MASSES

The common lateral discrete tumors or cysts of the neck are: branchial cysts, lymphomas, acute or chronic localized lymphadenitis and lipomas. Lipomas in this region are rather common and may be mistaken for more serious conditions. As elsewhere, they are smooth, multilobulated and usually fluctuant. Sometimes the characteristic lobulation can be demonstrated on x-ray films of the soft tissues.

BRANCHIOGENIC CYSTS.—There are several theories concerning the origin of branchiogenic cysts, but most of the evidence supports the belief that they arise from persistences of the second branchial cleft (Fig. 115, *B*). They are characteristically located along the anterior border of the sternocleidomastoid muscle at any level in the neck from the posterior belly of the digastric to the manubrium of the sternum. There may be a fistulous tract which extends up to the digastric muscle, arches medially behind the stylopharyngeus muscle and terminates in the tonsillar fossa. The cyst or tract is lined with ciliated and stratified squamous epithelium and contains a milky or mucoid material. An external fistula may be present. Treatment consists of complete surgical excision.

secondarily involved by processes affecting the superficial cervical nodes. The superior nodes of the deep cervical chain enter the jugular lymphatic trunks and form the inferior group below the bifurcation of the common carotid. Ultimately the channels join the thoracic duct on the left and the right lymph duct at the junction of the subclavian and internal jugular veins on the right.

A classification of the tumors and cysts of the neck (modified from Lahey) is given below. See also Figure 115, which shows some of the common masses of the neck.

MIDLINE MASSES:

Thyroglossal duct cyst (Fig. 115, *A*)	Adenoma of isthmus of thyroid
Pyramidal lobe of thyroid	Lingual goiter

LATERAL DISCRETE MASSES:

Lipoma	Discrete tuberculous node
Salivary gland tumors	Dermoid cyst
Discrete adenoma of thyroid	Carotid body tumor
Branchial cleft cyst (Fig. 115, *B*)	Localized acute or chronic cervical lymphadenitis
Secondary carcinoma	
Discrete lymphoma	

LATERAL MULTIPLE MASSES:

Acute or chronic lymphadenitis	Lymphomas
Nodular goiter	Tuberculous cervical adenitis
Metastatic cancer (Fig. 115, *C*)	Cystic hygroma (Fig. 115, *D*)

LOW MASSES EXTENDING INTO MEDIASTINUM:

Intrathoracic goiter
Metastatic cancer from abdomen or chest

MIDLINE MASSES OF THE NECK

Lesions in this location rarely present the difficult diagnostic problems seen in the lateral area. Midline masses are most often due to thyroglossal remnants or ectopic thyroid tissue. The pyramidal lobe, when present, extends upward from the isthmus, usually to the left of the thyroid cartilage. The only factor of clinical importance is that the pyramidal lobe should be removed during thyroidectomy. Lingual thyroid masses are very rare.

Because cysts in the neck are often very tense, the differentiation between cystic and solid tumors may be difficult. The tenseness of cysts and the mobility of the neck structures frequently makes fluctuation an unreliable sign of fluid. The location, mobility, fixation,

known, is notable for its slow and insidious onset and great chronicity. Although common years ago, it is rarely seen today. Tuberculous nodes become conglomerate, matted and ultimately undergo caseation necrosis. The overlying skin is bluish, and there may be painful fistulous tracts with characteristic ragged and undermined edges. The diagnosis is established by tuberculin testing, excision biopsy and guinea pig inoculation of the material aspirated from a caseous node. The measures which have proved useful in treating tuberculosis in other areas are also effective here. Radiotherapy and surgical excision are occasionally used.

METASTATIC DISEASE IN THE CERVICAL LYMPH NODES.—Gross lymphadenopathy from cancer results from a combination of a multiplication and aggregation of tumor cells and secondary inflammatory changes. Not every enlarged regional node draining a carcinomatous area contains cancer cells. The chief pathologic changes may be the result of inflammation. Cancer cells disseminate through lymphatic channels by embolism and spread from node to node in the direction of lymph flow. Reversal of lymph flow and retrograde spread of metastases occurs when lymphatic obstruction exists. Carcinomatous lymph nodes are often stony-hard and fused. Suppuration of nodes also occurs in advanced disease.

Metastatic cervical cancer is often the first sign of a primary malignancy elsewhere. The upper deep cervical nodes may be involved early in the course of cancer of the mouth or pharynx; the lower nodes of the deep cervical chain in primary malignant disease of the lung or the alimentary tract; and any of the deep nodes in cancer of the thyroid. All patients presenting discrete or multiple masses in the neck require special examinations of the oral cavity, the oropharynx and the nasopharynx. Proper equipment and lighting is necessary for this examination. Repeated examinations may be required before the primary tumor is discovered.

Where secondary spread to the neck has occurred, the primary tumor should be located, the diagnosis verified by biopsy and curative or palliative therapy applied as indicated.

MALIGNANT LYMPHOMAS.—This group includes: the giant follicle, the lymphocytic and the lymphoblastic lymphomas, Hodgkin's disease, reticulum cell sarcoma and their many biologic and histologic variants. The leukemias can be considered metastases in the blood from malignant lymphomas. The diagnosis of leukemia is established by studies of the blood, lymph nodes and bone marrow; the diagnosis of the

Carotid Body Tumor.—Tumors of the carotid body are rare, slow-growing and usually benign. They are single, solid lesions attached to or sometimes encircling the carotid bifurcation. They may be mistaken for carotid aneurysms. They lie high and deep in the neck and tend to extend upward, occasionally bulging into the pharynx. Because they are fixed to the carotid arteries, they cannot be dislocated downward. The carotid body syndrome is infrequently associated. Surgical treatment may be difficult and complicated. The tumor should be removed if continuity of the internal carotid can be preserved; otherwise the risk of cerebral ischemia with hemiplegia is great.

Aneurysm.—An aneurysm of the carotid artery or its branches is suggested by the presence of an expansile, pulsating mass which produces a thrill or a bruit. An arteriovenous aneurysm (fistula) is suggested by a history of a penetrating injury, a continuous machinery-like murmur and bradycardia when the fistula is occluded by pressure (Branham's sign).

Acute and Chronic Cervical Lymphadenitis.—In most cases of cervical lymphadenitis, several nodes will be enlarged. However, single masses are occasionally found. In the acute form of isolated cervical adenitis, a primary focus of infection in the skin, mouth, pharynx, sinuses, etc., can usually be found. Acute infections generally result from pyogenic organisms; chronic infections are more likely to be of the granulomatous type.

Lateral Multiple Masses

The most common conditions in this group are acute and chronic cervical lymphadenitis, metastatic carcinoma to the cervical nodes (Fig. 115, *C*), lymphoma, nodular goiter and cystic hygroma (Fig. 115, *D*).

Acute Cervical Lymphadenitis.—Inflammatory lymph nodes are usually the localized evidence of a staphylococcal or streptococcal infection Inflammatory nodes are multiple, tender, enlarged and often confluent. Suppuration may develop. The systemic signs of infection are present. The general principles for the treatment of infections apply in these conditions. Energetic treatment, directed at both the primary and secondary manifestations of the disease, often results in resolution without suppuration. If suppuration develops, the physician should avoid hasty incision for drainage.

Tuberculous Lymphadenitis.—"Scrofula," as it has long been

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lymphomas, by histologic examination of an accessible lymph node.

The lymphomas are most common in the middle and advanced age groups. The first complaint may be a single, deeply placed, firm and not freely movable mass in the upper part of the neck. Multiple nodal involvement occurs later. The nodes are sometimes fused together and there may be local tissue invasion, but suppuration is rare. Roentgen therapy is of value, but generally, surgical measures have little to offer.

In young adults the onset of Hodgkin's disease is often insidious, and the slow course may be deluding when considered in relation to the final fatal outcome. There is progressive and multiple involvement of the lymph nodes, which often begins in the cervical region. Hodgkin's disease may start as a unilateral lymphadenopathy, but generalized involvement of the lymph nodes is more common. The nodes are discrete, smooth, elastic and mobile.

The systemic signs of Hodgkin's disease are: weakness and malaise, anemia and fever of the Pel-Ebstein type; but diagnosis may be difficult because Hodgkin's disease often masquerades as other diseases The diagnosis is confirmed by biopsy. Roentgen therapy may hold the process in check for many years. Radical surgical treatment is ineffective.

Cystic Hygroma.—Cystic hygromas are formed by the sequestration of primitive lymphatic tissue of the jugular lymph sacs. In the embryo the jugular sacs are located in the neck near the junction of the internal jugular and subclavian veins. The cystic hygroma is most often found in infants or children, most often low in the neck, in the axillae or both areas. The tumor is a multiloculated, endothelial-lined, thin-walled, fluid-filled mass. The characteristic invasive property of primitive lymphatic tissue is noted in cystic hygroma. While the recommended treatment is complete surgical excision, this is not always practicable. In some instances, radiation or sclerosing solutions have been used with indifferent results.

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The thyroid is not generally affected by diseases which involve other tissues and organs. Acute thyroiditis, an uncommon acute inflammatory condition, is characterized by localized pain and tenderness and a systemic reaction which resembles that of an infectious disease. The process subsides spontaneously and operative treatment is not indicated.

In chronic thyroiditis there is a pronounced diffuse increase in the consistency of the gland, with varying degrees of enlargement and pressure symptoms. Three pathologic types which are difficult to differentiate clinically are: Riedel's struma (or "woody thyroiditis"), an iron-hard, fibroplastic goiter; Hashimoto's struma (struma lymphomatosa), with lymph follicles with germinal centers predominant; and Quervain's struma, a subacute giant cell thyroiditis. Surgery may be necessary to differentiate these conditions from carcinoma or to relieve pressure symptoms. Hypothyroidism may result from chronic thyroiditis when replacement of acinar tissue occurs.

A simple clinical classification of goiter is possible according to structure and function: (1) diffuse nontoxic goiter (colloid goiter); (2) nodular nontoxic goiter (adenomatous goiter); (3) nodular toxic goiter (adenomatous goiter with hyperthyroidism); and (4) diffuse toxic goiter (Graves' disease). The factors underlying the production of goiter are incompletely understood, but pathophysiologic changes resulting from acinar hyperplasia and involution are believed basic. Diffuse nontoxic, or colloid, goiter is thought to be the result of an iodine deficiency. A symmetrical and generalized enlargement of the gland results from excessive colloid.

Circumscribed single or multiple thyroid nodules are *common*. Variations in the histologic appearance of the nodules occur, and often evidence of both hyperplasia and involution may be seen, side by side. Degenerative changes in adenomata, such as cyst formation, hemorrhage, calcification or neoplasia, may be encountered. Localized areas of hyperfunction may produce all the systemic signs of primary hyperthyroidism. Solitary nodules (adenomas) are believed to possess malignant potentialities. Because the single thyroid nodule may be cancerous or may become cancerous, some authorities recommend excision of all solitary thyroid nodules.

The primary cause of thyrotoxicosis lies beyond the thyroid gland. Evidence suggests that functional neuroendocrine disturbances of the cerebral cortex and subcortical centers, the hypothalmus, the anterior

CHAPTER 26

The Thyroid Gland

THE THYROID gland develops from three primordia: the central portion of the gland from an invagination of the anterior wall of the primitive pharynx, and the lateral lobes from invagination of the fifth pharyngeal pouches. Residual tissue from midline anlage may remain as a pyramidal lobe, thyroglossal duct or cyst, or sublingual goiter. The thyroid gland is abundantly supplied with blood through the superior and inferior thyroid arteries. A fibrous capsule, formed by the enveloping deep cervical fascia, and a thin inner capsule cover the surface of the gland and provide a framework for the acinar tissue. The parathyroid glands, usually four in number, are yellowish brown structures closely resembling fat. They are located on the posterior surface of the thyroid adjacent to its main arterial branches. The recurrent laryngeal nerves generally lie in the tracheo-esophageal groove in close relationship with the inferior thyroid arteries, where they may be injured during operation. The recurrent, or inferior, nerves supply all the intrinsic muscles of the larynx except the cricothyroids, which are supplied by the superior laryngeals. The latter nerves are also sensory to the larynx.

Thyroid function is concerned with the processes of cellular oxidation, growth and development. Production of thyroid hormone (thyroxine) is regulated chiefly by the thyroid-stimulating hormone (TSH) of the anterior pituitary. Thyroid activity is frequently disturbed, and there is great variatio[illegible] thyroid occurs normall[illegible] and during pregnancy. [illegible] sible for the so-called "toxic goiter"; severe hypofunction is the basis for cretinism or myxedema. The condition of normality is known as the "euthyroid state."

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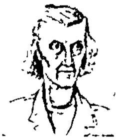

HYPERTHYROID

EUTHYROID

HYPOTHYROID

		BMR	PBI µg.%	I^{131} Uptake 4 hr	I^{131} Uptake 24 hr
Hyperthyroid	low	+25	10	50	60
	av.	+35	12	60	70
	high	+50	17	90	90
Euthyroid	low	−10	4	10	20
	av.	0	6	15	25
	high	+10	8	20	30
Hypothyroid	low	−40	0.3	0	0
	av.	−30	2.0	2	5
	high	−20	3.0	5	10

FIG. 116.—Usual laboratory findings in clinically hyperthyroid, euthyroid and hypothyroid patients. (From Evans, C. T.: J. Iowa M. Soc. 45:179, 1955.)

pituitary, the autonomic nervous system, the gonads and the adrenals are involved, but exact relationships are not known. Thyrotoxicosis has developed following severe emotional stress. Thyroid hormone in abnormal amounts is the basis for the signs and symptoms of thyrotoxicosis. Hyperfunction of the thyroid is associated with characteristic cytologic features: decreased stored acinar colloid, large columnar cells, increased vascularity and an increased number of lymphoid follicles.

The gross and histologic features of nodular toxic goiter are those of nodular nontoxic goiter plus added areas of hyperfunctioning thy-

roid tissue. Nodular toxic goiters usually contain areas of both hyperfunctioning and involuting tissue. Cystic nodules, areas of old and recent hemorrhage and calcific deposits are also common.

Compression of the trachea and/or esophagus or the possibility of malignant disease is an indication for operation for nontoxic goiter. Cosmetic considerations may also justify operative treatment. In general, it can be said that nodular goiter is a surgical disease.

Recently introduced technics for the clinical study of thyroid function include measurement of the protein-bound iodine level of serum and the uptake level of radioactive iodine by the gland. These tests of thyroid function and the basal metabolic rate (BMR) give three measures of thyroid activity. They are no more or no less accurate than the basal metabolic rate; but they deviate under different influences and, when properly interpreted, give valuable diagnostic and therapeutic information. Protein-bound iodine (PBI) is an index of the amount of iodine, united with protein, that is contained in body fluids; it represents the quantitative measurement of iodine, of thyroxin and related compounds circulating in the blood stream. If the patient has received neither iodine nor thyroid extract, the serum PBI concentration is an accurate measurement of the level of thyroid activity. The normal range of PBI is 3.5–8 μg./100 ml. of serum. Values above this figure indicate thyrotoxicosis; values below, hypothyroidism. The determination is meaningless if the patient has taken iodine-containing substances (cough syrup, x-ray contrast media, etc.) within weeks or months of the test.

The radioiodine (I^{131}) uptake depends on the amount of radioactive iodine which is stored in the thyroid over a definite period of time, and is correlated with the functional state of the gland and the iodide pool (Fig. 116). The amount of radioactivity can be determined by the use of a directional counting device. The normal range of uptake is 10–30 per cent in four hours and 20–40 per cent in twenty-four hours.

Clinical Findings

Four types of goiter are recognized clinically, as follows:

Diffuse Nontoxic Goiter.—Diffuse nontoxic goiter occurs predominantly in females during early adult life. The gland is uniformly enlarged, smooth and of normal consistency. The BMR is normal. Mild pressure symptoms or nodular goiter may develop. Surgical

treatment is sometimes indicated for cosmetic reasons. Iodine prevents the development of colloid goiter. It may also cause regression of the size of the gland.

NODULAR NONTOXIC GOITER.—There may be an isolated nodule (adenoma) in thyroid tissue, or diffuse nodularity with great variation in size and consistency of the separate nodules, but no toxic manifestations, although slight to severe pressure symptoms are common. Small, soft nodules may not always require removal, but those which are firm or enlarging should be treated surgically. Solitary nodules, especially in children and men, should be regarded with suspicion and removed because of the relatively high incidence of neoplastic changes in these masses. In most instances, carcinoma and adenomatous goiter cannot be distinguished by clinical signs alone.

NODULAR TOXIC GOITER.—In this condition the nodules are similar to those of nodular nontoxic goiter but they may be firmer and more fixed. They are encapsulated and show hyperplastic and involutional aggregations of tissue often associated with cystic, calcific or hemorrhagic degeneration. Occasionally, malignant changes are found. The symptoms of toxicity are generally delayed, often for ten to fifteen years, and are milder than those of diffuse goiter. Slow progression and insidious changes may result in cardiac decompensation with auricular fibrillation (thyrocardiac). Pressure symptoms, especially in the intrathoracic substernal type, are common. Intrathoracic goiter is usually nontoxic. The treatment of nodular goiter is primarily surgical; but, if necessary, antithyroid drugs and iodine can be used before operation, in order to achieve a euthyroid state.

DIFFUSE TOXIC GOITER.—Diffuse toxic goiter has no relation to endemic goiter. It is thought that some stimulus produces thyroid hyperplasia with excessive production of thyroid hormone. The onset of thyrotoxicosis may be associated with a severe emotional disturbance, certain infectious diseases or extreme fatigue. Females are most commonly afflicted in early adult life.

In diffuse toxic goiter the gland is smooth, firm and sometimes tender. It may or may not be enlarged. Increased circulation may be associated with a thrill or bruit over the thyroid.

The clinical course of diffuse toxic goiter is marked by exacerbations and remissions. Every cell and all body systems may be affected. The most outstanding clinical changes are: nervousness, tremor and emotional instability; weight loss in spite of excessive food intake;

muscular weakness; tachycardia, dyspnea and other cardiac manifestations; intolerance to heat; excessive sweating; and the eye signs of goiter, including exophthalmos, widening of the palpebral fissure, lid lag and inability to converge.

Medical Treatment and Radiation in Thyroid Disease

Although not always practicable, treatment for the patient with thyroid disease is best determined by a combined medical group, consisting of internist, surgeon and radiotherapist. Surgical treatment once constituted the only proved method for the treatment of goiter. Now, other effective measures are widely used. The antithyroid drugs and radioiodine have had extensive clinical use. Nodular goiter continues to be primarily a surgical problem. Toxic diffuse goiter may be treated either by operation or by the use of radioiodine. Antithyroid drugs are used in selected cases, but recurrent hyperthyroidism has been observed in about 50 per cent of cases when the drugs are discontinued. At the present time, all methods of treatment are directed at the target organ, the thyroid gland. In the preoperative preparation, thiouracil derivatives and iodine are the basic drugs.

The thiouracil compounds are most useful for preoperative, rather than definitive, treatment. They block the combination of iodine and tyrosine which normally forms di-iodotyrosine in the acinar cells. Associated with the blocking effect, there is an intensification of the thyroid stimulating action of the anterior pituitary (TSH) on the thyroid cells. The effect of thiouracil and related drugs on the thyroid histologically is the opposite to that of iodine. These drugs produce an increase in all aspects of hyperplasia, including an increase in the number of mitotic figures, an increase in papillary infoldings, a decrease in the amount of stored colloid, an increase in lymphoid tissue and increased vascularity. Iodine will decrease or inhibit the hyperplastic effect of thiouracil. Advantage of this fact is taken in preoperative preparation, when iodine is administered for a short period, usually seven to fourteen days, to make the gland less vascular and to make operation easier.

The thiourea derivatives (thiouracil, propylthiouracil and Tapazole®) are rapidly excreted and therefore must be given at regular intervals in order to maintain therapeutic levels. When there is intense hyperplasia and little stored colloid, the BMR drops faster than when

histologic changes are less striking. Iodine produces a rapid, but often incomplete and brief, remission of thyrotoxicosis. It causes a reduction of thyroid hyperplasia with the storage of colloid, and it prevents thyroxin secretion for a short period of time. Iodine has long been used in preoperative treatment of thyrotoxicosis.

Radioiodine is taken up by the thyroid cells, which then are subjected to radiation. The effect is produced by high-energy beta radiation with a relatively short penetration of not more than 2–3 mm. The possibility of late neoplastic changes due to such radiation has not been ruled out, although the hazard would not seem to be great. The more hyperplastic the goiter, the more radiosensitive it is. Nodular goiters are often more resistant. Radioiodine can be given in a single dose or in multiple doses. About three months is required for the full radiation effect to become apparent. Radioiodine finds its greatest usefulness in the treatment of selected patients with diffuse toxic goiter, recurrent hyperthyroidism, thyrocardiac disease, severe exophthalmos and differentiated forms of thyroid cancer and in the treatment of those who are poor operative risks.

The basic objective of preoperative management is to bring the patient to a euthyroid state and to reduce the risk of surgical treatment to a minimum.

The reduction in metabolic rate is achieved by the administration of antithyroid drugs (e.g., propylthiouracil in divided doses of 300–600 mg./day for three to ten weeks). Ten days to two weeks before operation, iodine therapy is given to produce involution and decreased vascularity of the thyroid (Lugol's solution, 0.6 ml. three times a day)

A high-calorie diet, rich in carbohydrate and high-quality protein, supplemented with vitamins (especially B complex) in adequate amounts is prescribed.

As the antithyroid drugs induce a remission, the pulse rate will show a significant reduction and other signs of toxicity will diminish. The thiouracil drugs occasionally produce complications, including agranulocytosis, fever, leukopenia, nausea and vomiting, and dermatitis. They should be used with care. Physical and mental rest, sedatives if required, and frequent return visits are in order. Under such treatment the decrease in the BMR will average above 1 per cent a day. Thus, a patient with a BMR of plus 60 will require about sixty days of treatment to reach a euthyroid state.

Surgical Treatment of Hyperthyroidism

Surgical management consists in preparation until the euthyroid state is realized, proper selection and administration of anesthesia, meticulous surgical technic and good judgment as to the amount of thyroid to be removed, an appreciation of the operative hazards and knowledge of how to avoid them, and a determination to continue care until the patient is restored to health.

Thyroidectomy for hyperthyroidism is a well-established operation. Only a small segment of each thyroid lobe is preserved. The amount of thyroid tissue preserved is a matter of nice judgment: resection of too much is followed by hypothyroidism; of too little, by hyperplasia of the remaining tissue and recurrent hyperthyroidism. Care must be taken to avoid injury to the parathyroid glands and the recurrent laryngeal nerves. Many surgeons demonstrate the nerves at operation, believing that "if you can see them, you will not hurt them"; while others prefer to guard against injury by avoiding the area where the nerves are normally located.

Unusual vascularity of the thyroid or inadequate control of bleeding may lead to serious operative or postoperative trouble. Attention to hemostasis beyond that usually required in other operations is essential; for the neck offers little resistance to continued bleeding, and the dangers of asphyxia from tracheal compression are very real.

Postoperative Care

In the postoperative care, the following factors are important:

1. The relief of pain usually calls for opiates, as needed. The barbiturates and paraldehyde may be used for supplementary sedation.

2. The fluid intake must be adequate. Usually the patient is able to take oral fluids soon after operation. The diet should be increased as tolerated.

3. The semisitting position in bed is most comfortable. Ambulation should be started the day after operation.

4. The dressing should not be applied tightly. If drains are used, they should usually be removed after twenty-four hours.

5. Oxygen, if necessary, should be given by nasal catheter. Fever and an unusually rapid heart rate may call for an oxygen tent, which

provides a cool and comfortable environment for the dyspneic and restless patient.

6. Antithyroid therapy is rarely needed postoperatively.

Postoperative Complications

The outstanding postoperative complications follow:

Postoperative Hemorrhage.—Hemorrhage should be suspected if sudden dyspnea, cyanosis and collapse occur. There may be tracheal compression by a hematoma. Prompt action is necessary if the patient is to survive. The danger of asphyxia, rather than blood loss, is the major and immediate concern. Hemorrhage may be rapid, e.g., owing to slippage of an arterial ligature, or slow and progressive if from many small vessels. Venous bleeding may be extensive and can also cause acute respiratory embarrassment. The dressings should be removed and the wound opened without delay. While aseptic conditions should be used if possible, it should be realized that this is an emergency condition and that delay is dangerous. Precious time should not be wasted by moving the patient to the operating room if there is impending airway obstruction. Instead, a sterile forceps or a gloved finger should be introduced and the hematoma evacuated. Bleeding is usually controlled by packing with hemostatic substance or gauze. Secondary ligation of vessels is necessary in hemorrhage from major arteries. Unless prompt relief of a tracheal obstruction occurs, intubation or tracheostomy is required without delay.

Recurrent Laryngeal Nerve Injury.—Unilateral injury results in immobility of the cord, usually in the midline position. The voice may be weak or hoarse. If voice changes are noted immediately after operation, nerve damage is probable and may be permanent; if voice changes develop later, edema is the most probable cause and return of function is likely. In unilateral nerve injury there is no specific method of treatment.

Bilateral nerve paralysis is an uncommon but serious complication. The cords may be fixed in the midline, so as to produce narrowing of the rima glottidis, with stridor and signs of asphyxia. The patient is unable to speak. Edema of the larynx may accentuate the difficulty and force immediate tracheostomy. In some cases, respiration can be supported by an oxygen-helium mixture, but tracheostomy must not be postponed if obstruction of the airway persists.

Bilateral nerve paralysis may sometimes lead to late respiratory

obstruction, which appears after a period of several months as contracture occurs and the cords approach the midline. "Crowing" breathing and marked dyspnea on exertion then occur. Operative procedures directed toward widening the rima, such as an arytenoid excision, then become necessary.

POSTOPERATIVE PARATHYROID TETANY.—Operative injury to the parathyroids, with edema and ischemia, or occasionally accidental removal *results in symptoms of tetany due to hypocalcemia in from* twelve to forty-eight hours. The symptoms are due to increased neuromuscular irritability and consist of restlessness, irritability, paresthesias of the extremities, pain and spasm of muscles and tachycardia. Examination reveals twitching of the upper lip when the facial nerve is stimulated by tapping (Chvostek's sign) and carpopedal spasm that is aggravated by constricting the arm with a blood pressure cuff (Trousseau's sign). The serum calcium is below the normal level of 8.5 mg./100 ml.; the serum phosphorous is elevated above the normal level of 5 mg./100 ml. No calcium is excreted in the urine (Sulkowitch's test). The laryngospasm of tetany must be differentiated from obstructive dyspnea due to other causes, such as recurrent nerve paralysis, tracheal collapse or postoperative hemorrhage, or edema.

Intravenous calcium, lactate or gluconate will relieve the acute symptoms. It is well to draw blood for calcium and phosphorus determinations before calcium is administered, in order to confirm the diagnosis. In most cases, parathyroid tetany is temporary. If chronic tetany develops, carefully planned management includes: restricted activity, a high-calcium and low-phosphorus diet, vitamin D and, infrequently, dihydrotachysterol (A. T. 10). Because of antihormone effects, parathyroid extract will relieve symptoms only for short periods. It is rarely used.

MALIGNANT EXOPHTHALMOS.—Exophthalmos may regress, remain stationary or increase after operation. When progressive exophthalmos develops and vision is threatened, operative decompression for the relief of intra-orbital tension must be considered.

THYROID STORM.—Postoperative toxic crises do not occur when the patients have been adequately prepared and are in a euthyroid state at the time of operation. The complication of toxic crises is a serious and sometimes fatal accentuation of hyperthyroidism occurring after operation. While formerly common, it is now rarely seen. There is nervousness, restlessness, nausea and vomiting, diarrhea and marked

tachycardia. The patient becomes delirious and unmanageable. The body temperature rises rapidly to 105° F. or higher. Death is due to cardiovascular collapse or hepatic failure. Supportive measures include: sponging, cooling enemas, sedatives, morphine, oxygen, intravenous dextrose, iodine and ACTH or cortisone.

POSTOPERATIVE MYXEDEMA.—Hypothyroidism of varying degree follows excessive surgical removal of the thyroid or I^{131} overdosage. Thyroid substance is given as replacement therapy. Such management is indicated after radical operations for thyroid cancer.

RECURRENT TOXIC GOITER.—Toxic goiter may recur when insufficient thyroid tissue has been removed. Under these circumstances, I^{131} administration should be considered because there is a relatively higher incidence of complications following secondary operations, as compared to primary operations.

CARCINOMA OF THE THYROID

The subject of thyroid cancer has received increasing attention in recent years. Much of the discussion has concerned such issues as the relation of isolated nodules of the thyroid to the pathogenesis of carcinoma, the problems of clinical and pathologic diagnosis, the unexplained variations in the clinical behavior of thyroid tumors and the indications for, and application of, surgical, radiation and hormonal alteration therapy in the management of this disease. Some of these issues will be considered in this discussion.

Cancer of the thyroid occurs at all ages. The mean age in a group of patients studied at the University Hospitals of the State University of Iowa was 46.7 years. Some of the cases were seen in childhood and early adult life. In the childhood group, one out of three masses in the thyroid gland proved to be malignant.

While the incidence of goiter (both toxic and nontoxic) is seven to one in favor of females, the incidence of thyroid cancer is about the same in men and women. It is apparent, therefore, that the odds in favor of a mass in the thyroid being cancer are greatest in children and men.

Many writers have emphasized that the single nodule of the thyroid is most likely to be or to become malignant. While this may be true, it does not mean that multinodular goiters are necessarily benign. Glands which are the site of diffuse adenomatous changes may also be malignant. Of some importance to clinical diagnosis is

the fact that cancer is almost never associated with diffuse toxic goiter; also, it is rare in nodular toxic goiter.

PATHOLOGY.—The following classification of thyroid cancer, which has been suggested by Warren and Meissner, is based on pathologic findings and correlated, to some extent, with biologic behavior, response to treatment and prognosis:

Differentiated tumors	Undifferentiated tumors
Follicular carcinoma	Small cell carcinoma
Papillary carcinoma	Giant cell carcinoma

Miscellaneous tumors
- Epidermoid carcinoma
- Hurthle cell carcinoma
- Fibrosarcoma

The differentiated tumors are most common in young people (before age 40) and often follow a relatively prolonged course. Some of them elaborate colloid and show an avidity for iodine. They are usually more amenable to treatment and have a better prognosis than the undifferentiated tumors.

The undifferentiated tumors tend to occur with greatest frequency after the age of 40. They produce no colloid, grow rapidly and invade diffusely. The cure rate is low, but prolonged survival with the disease is common. Epidermoid cancers of the thyroid are very rare and highly malignant. Hürthle cell cancer is uncommon but not rare. It presents an unusual histologic picture of altered epithelial cells resembling liver parenchyma and behaves more like the differentiated tumors. Fibrosarcoma of the thyroid is very rare.

The histopathology of thyroid cancer shows great variation. Papillary, follicular and structureless areas (sheets of cells) are often found in different areas of the same tumor. The usual histologic criteria of malignancy may not be found on the microscopic sections, and yet the behavior of the tumor indicates it to be a cancer. The finding of venous invasion is sometimes the only criterion of a malignant process. In some patients, capsular invasion or the presence of metastases in cervical nodes may be the only indication that the mass is in fact a cancer. In still others, tumor cells may be found in cervical nodes in the absence of a discernible primary tumor in the thyroid itself. This condition, previously called "lateral aberrant thyroid," is believed to represent metastases from a microscopic primary tumor of the homolateral thyroid lobe.

SYMPTOMS AND SIGNS.—There are no early signs of cancer of the thyroid beyond the presence of a nodule or nodules or of enlarged

cervical nodes As in all other types of malignant disease, pain is a late symptom. As the growth progresses, there may be a noticeable increase in the size of the mass and fixation to the neck muscles and hollow structures. Tightness and difficulty in swallowing and in breathing may be observed by the patient. Later, there will be definite dyspnea, dysphagia, recurrent nerve paralysis with hoarseness, marked

FIG. 117.—Carcinoma of the thyroid gland. Note the extensive involvement The patient also had local and distant metastases.

induration of the neck and perhaps cervical, pulmonary or bone metastases.

The cancerous thyroid gland (Fig. 117) is usually irregularly enlarged, hard, nontender, nodular (either single or multiple) and somewhat fixed to the surrounding structures. Similar changes may be found in chronic thyroiditis, in hemorrhage into an adenoma or in advanced benign nodular goiter with degeneration. When the patient gives a history of a rapid increase in the size of a thyroid nodule (i.e., within a matter of hours or days), the most likely explanation is that a spontaneous hemorrhage has occurred into the nodule, but this phenomenon can occur in both benign or malignant tumors.

Thyroid cancer produces both lymphatic and blood borne metastases. There may be a noteworthy discrepancy between the known duration, size and histologic picture and the extent of the

metastases. Small and benign-appearing tumors sometimes produce widespread dissemination, while large, infiltrating and vicious-appearing tumors remain confined to the neck. This points up the need for a careful search for metastases in patients in whom the diagnosis of thyroid cancer is entertained.

DIAGNOSIS.—The diagnosis of cancer of the thyroid should be considered in all patients with a mass in the thyroid or enlarged lateral cervical lymph nodes which cannot be ascribed to other causes. Thus the various local and systemic causes of masses in the neck should be ruled out. An x-ray film of the chest is in order, to determine the presence of mediastinal, pulmonary or bone extension. A complete x-ray survey of the skeleton may also be indicated. Thyroid function studies are generally of little value in differential diagnosis. The patient is usually euthyroid, and there is likely to be little I^{131} uptake by the tumor; but occasionally the thyroid nodule has sufficient avidity for iodine to indicate the possible effectiveness of I^{131} in the control of metastases or recurrences, should the nodule prove to be malignant.

Needle aspiration biopsy of the thyroid has definite limitations and, except in expert hands, is likely to do more harm than good. Unless this procedure leads to positive diagnosis, it is not helpful. It would seem, therefore, that definitive diagnosis is best established by operation and examination of the excised tissue. It is a fundamental rule that one must avoid incising the tumor or breaking its capsule, lest dissemination, or "seeding," of neoplastic cells into the operative field occurs. To avoid such a possibility, the entire involved thyroid lobe is usually removed.

TREATMENT.—Of the several modalities available for the treatment of thyroid cancer, surgical extirpation remains the most promising. In certain localized carcinomas, complete removal of the involved lobe (lobectomy) may be adequate. This problem often arises when an operation has been undertaken for a benign adenoma of the thyroid which is subsequently found to be thyroid cancer in the fixed-tissue sections. When the diagnosis of operable thyroid cancer is established preoperatively or at operation, total thyroidectomy and homolateral neck dissection must be considered. For bilateral cervical node involvement, simultaneous or staged neck dissection with total thyroidectomy has been done. In certain good-risk patients, both cervical and superior mediastinal node dissection has been recommended.

Surgical treatment of undifferentiated thyroid cancers, which comprise about 20 per cent of all thyroid cancers, is less predictable and often ineffective. Although some patients appear incurable at the time they are first examined, they may be benefited by operation. An attempt should be made to remove the tumor and relieve impending or existent obstruction to the neck structures. Occasionally, simple

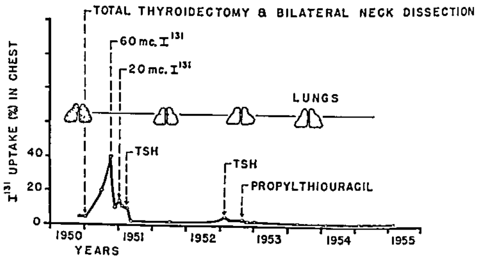

FIG. 118.—Results of I^{131} therapy of thyroid carcinoma. Surveys before surgery indicated usual uptake in the thyroid region, with only very slight uptake in the lung metastases. Histopathologic examination revealed the carcinoma to be mostly papillary but with some follicle formation. A few of the follicles accumulated I^{131}, as indicated from radioautographic studies Within a few months after removal of the thyroid, the metastases in the lungs accumulated I^{131} to a satisfactory degree, and I^{131} therapy was then instituted Within a year, the iodine-accumulating cells were destroyed, and attempts to stimulate further activity with TSH and with propylthiouracil were fruitless Consequently, therapy was discontinued. As the patient has become myxedematous, thyroid extract is administered except when I^{131} tests are done (twice a year) to determine whether further treatment is necessary. So far, there have been no indications, either from I^{131} studies or from clinical findings, that carcinoma persists. (From Evans, T. C. J Iowa M. Soc. 45:182, 1955)

division of the thyroid isthmus may give a measure of relief. In the later stages of the disease, tracheostomy is often required.

Radiotherapy is also used in the management of patients with thyroid cancer External radiation is valuable for the control of local extension in the neck and bone metastases. Radioiodine provides a means for effective control of metastases (Fig. 118) in a small percentage of patients. The high hopes which were held by workers in this field for the radioactive substances have not materialized because so few thyroid tumors exhibit an avidity for iodine. Despite this,

however, some striking results have been achieved, and patients should not be deprived of the possible benefits of I^{131} when the situation is such as to suggest it may be helpful. The iodine uptake in some differentiated tumors can be increased by making the patient hypothyroid. This is accomplished by performing total thyroidectomy (which is frequently done for thyroid cancer), by giving antithyroid drugs or by giving TSH. In each instance, an increase in circulating TSH (either from endogenous or exogenous sources) results in stimulation of the metastases to produce colloid and to increase the uptake of I^{131}. Thus a *local radiation effect can be achieved.*

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CHAPTER 27

The Breast

It is wise to accept with humility that however experienced we are we cannot differentiate with certainty on clinical grounds alone between non-malignant and early malignant breast disease. . . . We must become more and more accustomed to suspecting malignant disease on less and less clinical evidence.—RIDDELL (1948).*

DISEASES OF the female breast are very common in clinical practice. Malignant and benign tumors, cystic mastitis and functional complaints comprise the majority of conditions encountered. Of these, the most important is cancer. It is the predominant malignant tumor of the female, in the United States accounting for a high incidence of morbidity and over 20,000 deaths yearly. Today, the main hope for reducing this high toll lies in early detection and treatment of all breast masses. To this end there is a need for continued public emphasis on the importance of self-examination of the breast and prompt medical attention for all abnormalities so discovered.

The problems which arise in connection with breast diseases in general, and breast cancer in particular, encompass nearly the entire field of neoplasia. From a teaching viewpoint, breast cancer is to surgery what typhoid fever was to medicine a few generations ago. Just as Osler, in his *Textbook of Medicine,* described many aspects of typhoid which were applicable to the entire field of medicine; so a thorough understanding of breast cancer and its many ramifications will serve the student as a foundation for the study of other surgical conditions.

*Riddell, V.: Early diagnosis and treatment of carcinoma of the breast, Brit. M. J. 2:635, 1948.

GENERAL CONSIDERATIONS

The breast is a modified sebaceous gland composed of fifteen to twenty branching duct systems, each opening independently at the nipple. From each main duct, branching lobular side ducts and ductules communicate with the glandular tissue (acini). A fibrous network (Cooper's suspensory ligaments) and a varying amount of fat give the breast support and form.

The female breast is a hemispherical organ extending from the second to the sixth ribs vertically and from the parasternal to the midaxillary line horizontally. A triangular segment which extends toward the axilla (axillary tail of Spence) provides a "tear drop," rather than a round, distribution of breast tissue on the chest wall. The centrally located nipple, in which tiny duct openings may sometimes be seen, projects at right angles from the pigmented areola. Scattered throughout the areola are many small protuberances, called the glands of Montgomery, which serve to lubricate the nipple during lactation.

The breast is richly supplied with blood vessels. The chief arterial supply comes from (1) the lateral thoracic artery from the axillary artery, (2) the anterior perforating intercostals from the internal mammary artery and (3) the lateral perforating intercostals from the corresponding intercostal arteries. The pattern of venous drainage is, in general, similar to that of the arterial supply.

The lymphatics of the breast originate in the glandular tissue and pass in the interlobular supporting tissue to the superficial lymphatic plexuses of the skin and areola and to the deep lymphatic network on the fascial planes of the muscles and chest wall. From these channels, lymph is conveyed via communicating vessels (Fig. 119) to lymph node aggregations located in the axilla and the anterior mediastinum (internal mammary nodes). The lymphatics of the skin of the breast form a continuous network with the lymphatics of the skin of the surrounding region—i.e., the chest, neck, abdomen and the opposite breast. Blockage of the primary routes of lymphatic drainage causes shunting of lymph to other areas, including the supraclavicular and cervical nodes, the node between the pectoral muscles (Rotter's node), the opposite breast or axillae, or the rectus sheath, falciform ligament and peritoneal cavity.

The number and distribution of the axillary nodes is such that they serve as a relatively efficient lymph filtering system. The same

is not true of the internal mammary nodes. This group consists of only three to five small nodes, arranged in tandem and inaccessible except on opening the chest wall.

Lymphatic drainage from the outer half of the breast and areola is predominantly to the axillary nodes. The inner half drains to either

Fig 119.—The lymph drainage of the breast. Note the two important primary routes: to the axillary nodes and to the internal mammary nodes. Direct drainage to the supraclavicular fossa, the opposite breast and the lymphatics of the rectus sheath and abdomen usually occurs late in breast cancer

(or both) the axillary and internal mammary node areas. It should therefore be apparent that the routes of lymph drainage of the breast, and likewise those of dissemination of breast cancer through the lymphatics, is subject to variation and often unpredictable.

The noticeable morphologic and physiologic alterations of the breast which are controlled by the endocrine system (pituitary, ovarian and adrenal) can be correlated with the sex life of the individual. In the young of both sexes, the breasts consist of simple branching ducts without acini. In the female at puberty there is

branching of the duct systems and formation of some acini. The male breast remains almost unchanged throughout life.

The onset of menstruation, with ovulation and formation of the corpus luteum, is associated with growth of the glandular tissue. In the absence of impregnation, involution of the breasts occurs. These changes are repeated with each menstrual cycle. Should impregnation occur, extreme hyperplasia of the glandular and duct tissue follows. Droplets of colostrum appear in the acini; and, following childbirth, milk is produced. If lactation is inhibited, involution of the breast tissue follows. After the menopause, atrophy of the glandular, ductular and supporting tissues occurs. Although the epithelial changes at this time are predominantly those of involution, varying degrees of hyperplasia are also found.

EXAMINATION

The history which the patient gives may suggest the nature of the breast disturbance. For example: acute pain and swelling which develop during lactation are probably due to infection and abscess; cyclic pain and swelling are common in cystic mastitis; a serosanguineous nipple discharge suggests a lesion of the duct system; a tumor which follows a severe blow to the breast may be an area of traumatic fat necrosis; an isolated mass in a very young woman is likely to be a fibroadenoma; and a hard asymptomatic mass at any adult age is possibly cancer. While the history is an aid to diagnosis, the physician often gives too much weight to it, to the exclusion of other findings.

The following points should be established:

1. Is there a dominant lump in the breast? If so, how was it discovered? What symptoms, if any, changes in size; and relation to the menstrual cycle? Presence of a nipple discharge?
2. Has there been an injury to the breast? Any infections, operations or previous breast tumors? Record pregnancy and lactation history.
3. Is there a family history of breast cancer?
4. Does the patient have symptoms suggesting metastases to the skeleton, lungs, cranium, etc. If so, these areas must be investigated.

The examination (Fig. 120) should be conducted in a well-lighted room with a nurse in attendance, if possible. It should be done in an

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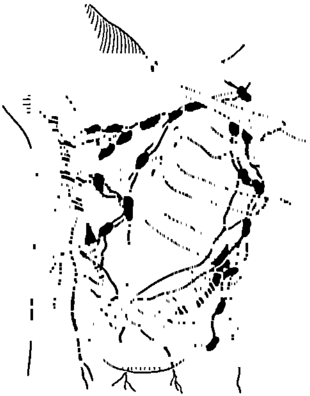

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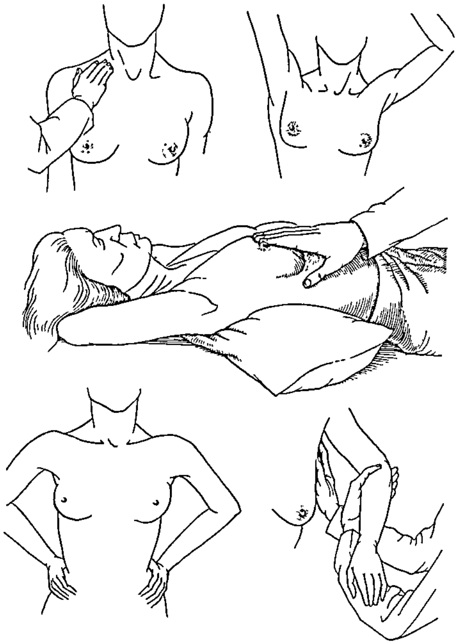

FIG 120.—Steps in the examination of the breast.

unhurried, systematic manner, with due regard for the patient's modesty and anxiety. During the examination the physician may avail himself of the opportunity to explain and demonstrate self-examination of the breast.

First, the patient should sit with the chest exposed, facing the examiner. The breasts are inspected and compared for symmetry, contour, color, appearance of the nipples and areolae, distribution of the superficial veins and changes in the skin.

Next the patient is requested to "put your arms straight above your head." The examiner again looks for changes in symmetry and contour. Then the patient is asked to "place your hands on your hips and push down." This maneuver causes contraction of the pectoral muscles and may elicit or accentuate deformities, particularly a "dimpling" sign. The patient is now asked to "lean forward with your arms in front of you." The examiner supports the patient's arms and inspects the breasts as they hang at an angle from the chest wall. Previous unobserved alterations are often detected with the maneuver.

The nipples and areolae are then palpated and compressed for fixation or discharge. Bimanual palpation of the breasts may also be performed.

The axillae are examined with the patient still in the sitting position. The examiner supports the patient's forearm with one hand while he palpates with his other hand. The patient's arm and shoulder should be relaxed and under control of the examiner. The arm is moved to gain access to all portions of the axilla. Normally, no nodes will be palpated, but small soft nodes are not necessarily abnormal. One should compare the findings on the two sides before drawing conclusions.

The patient is next placed on the examining table with a small pillow under one shoulder. The pillow should elevate the shoulder enough to rotate the breast inward or "pancake" it on the chest wall. This maneuver places a relatively thin layer of breast tissue over a wide area of chest wall and facilitates inspection and palpation. All sectors of the breast are reviewed, compressing the breast tissue against the chest wall. The lateral half, including the axillary tail, is first examined; then, with the patient's arm extended above the head to tense the underlying fascia, the medial half is examined.

Normally, the breast presents an even consistency, similar to the subcutaneous fat of an obese person. There may be some diffuse nodularity, according to the distribution of fat and fibrous and paren-

chymal elements. A normal finding which is a frequent cause for confusion is the condensation of fibroareolar tissue inferiorly, where the breast becomes confluent with the chest wall.

If there is discharge from the nipple, a smear should be made for microscopic examination. It should be studied for red cells, pus cells and exfoliated epithelium. When the smear is stained according to the Papanicolaou technic, the clinical diagnosis of cancer will occasionally be reinforced by a finding of abnormal epithelial cells.

Transillumination of a breast mass in a darkroom is an aid to differentiation of cystic and solid masses. X-ray films for soft-tissue detail and infra-red photographs for comparison of the superficial veins of the breasts, are interesting, but rarely helpful, investigative procedures.

Roentgenography of the chest should be routine when the diagnosis of breast cancer is considered. In the event that the clinical findings suggest the possibility of distant metastases, survey films of the spine, pelvis and skull are also in order. It should be pointed out that inability to demonstrate bone metastases on the x-ray films does not necessarily exclude the presence of metastases.

CLINICAL CONDITIONS

Cystic Disease

Cystic disease (cystic mastitis, mammary dysplasia) is a common breast condition which is believed to be the result of an endocrine imbalance or an abnormal response to endocrine stimulation. It occurs in microscopic or gross form in many supposedly normal patients, in whom it may be considered a physiologic, rather than a pathologic, condition. Pathologically, cystic disease is characterized by epithelial hyperplasia, cyst formation and interstitial fibrosis. One or more of these changes are usually prominent, but in nearly all specimens the other changes can also be demonstrated. The alterations in epithelium and the fibrous tissue are probably the result of an exaggerated hyperplasia-involution process.

Cystic disease occurs in both nulliparous and parous women, who are usually between 30 and 50 years of age. Some patients complain of pain in the breasts which is most severe during the premenstrual phase of the cycle. The pain may be localized or diffuse. Sometimes the pain radiates into the shoulder or arm, where it is quite disabling.

Associated swelling and tenderness of the breasts are common. Other patients complain simply of a mass or masses in the breast; and still others have no subjective complaints, but the breasts are found to be nodular, hard or ropy. These changes are considered a part of the same process.

The clinical findings are variable. Both breasts may be diffusely shotty or granular, or there may be isolated areas of lobular thickening or cyst formation. Sometimes a single round, isolated mass which is said to vary in size with the menstrual cycle is found. The examiner may find several other masses (cysts) not previously observed by the patient. The cysts may be discrete or conglomerate, soft or hard, tender or nontender. There is no tendency to skin fixation or retraction, and the axillae are generally negative. Nipple discharge is unusual.

Cystic disease is commonly the source of great discomfort and frequently cancerphobia. Although the risk of cancer development in patients with cystic disease is probably not significantly greater than it is for women with normal breasts, the high incidence of cancer in this age group is such as to warrant biopsy in the majority of cases.

Excision of the breast cyst or the area of localized induration is usually adequate. The excised tissue should be immediately examined by a pathologist. Rarely will there be found an unusual degree of epithelial activity which fulfils all the criteria of a malignant condition except for invasion. Some pathologists regard such changes as premalignant. Under these circumstances, and particularly if there is a strong family history of breast cancer, simple excision of the breasts must be considered.

After the diagnosis of cystic disease has been established, the patient should be re-examined periodically and treated symptomatically. If necessary, an uplift brassiere should be secured. During periods when the pain is severe, hot (or cold) applications, analgesics and rest are indicated. Aspiration of recurrent breast cysts is recommended. Generally, hormone preparations give only transitory relief and may be dangerous. They should not be used. With the menopause the symptoms tend to regress.

Fibroadenoma

The fibroadenoma occurs predominantly in young women (20–35 years of age). Typically, it is a nontender, isolated, freely movable

and "poppable" marble-like mass. It arises from the ducts and contains both fibrous and epithelial elements, as the name indicates. Fibroadenomas vary in size from a few millimeters to several centimeters. Local excision is adequate treatment, but new primary tumors may appear. Sarcomatous degeneration is unusual but leads to a large, nodular type of tumor known as "cystosarcoma phyllodes."

Duct Papilloma

Duct papillomas are most often encountered during the menopause Often, they cause a serous or serosanguineous nipple discharge. They generally arise in the terminal ducts near the nipple or areolae; and they tend to be small, soft, single or multiple growths with frond-like projections. Frequently, they cannot be located by palpation alone Under these conditions, if the various sectors of the breast are tested by applying point pressure, the appearance of a nipple discharge will indicate the probable location of the papilloma. Simple excision is adequate unless the tumor is found to be malignant (papillary carcinoma).

Traumatic Fat Necrosis

Traumatic fat necrosis should be suspected when a fixed mass appears in the breast shortly after the patient has sustained a significant injury. Often the patient is one who has large and pendulous breasts. The injury may be the result of having been thrown against a steering wheel. Immediately following the injury there is pain, swelling and a "black and blue" area. Later, a hard irregular mass appears. The mass consists of saponified fat liberated from devitalized fat cells, the products of inflammation and calcium soaps. The clinical signs are often strongly suggestive of carcinoma, and excisional biopsy is necessary for diagnosis.

Plasma Cell Mastitis

An uncommon benign lesion of the breast, which also may bear a close resemblance to cancer, is plasma cell mastitis. It usually occurs in parous women and may be associated with chronic nipple discharge. The discharge is serous or purulent, rarely bloody. The breast is nodular, thickened, irregular and may contain a hard, fixed

mass. The presence of enlarged axillary nodes may further support the clinical diagnosis of cancer. The diagnosis is established by biopsy.

Plasma cell mastitis is probably caused by the irritant effect of retained breast secretion, especially the lipid fraction. There is dilatation and atrophy of the ducts, with a varying degree of stromal reaction, coincident with the appearance of the irritant material beyond the ducts. Large numbers of plasma cells are found in these areas. Haagensen has suggested that the condition be called "mammary duct ectasia" in keeping with its pathologic features.

Breast Abscess

Acute infections of the breast are unusual except during pregnancy and lactation. The principles of treatment are those which are common to all infections. They include: localization of the infection; dependent surgical drainage, when indicated; antibacterial therapy; and closure of the defect, when necessary. Chronic infections sometimes result from inadequate treatment of acute infections and rarely are caused by organisms of the chronic granuloma group. Tuberculosis of the breast is generally secondary to a pulmonary infection.

It is necessary to consider the possibility of a breast cancer (inflammatory type) masquerading as a breast infection when a portal of entry for bacteria cannot be demonstrated and when a mass which could represent a deep abscess appears in the breast during pregnancy or lactation. Under both conditions the diagnosis must be established by biopsy.

Cancer

Breast cancer constitutes about 25 per cent of all cancer arising in women and about 18 per cent of all deaths from cancer in women; its peak incidence occurs between the ages of 40 and 70 years, with the mean age of onset being 57 years. It does not occur in the prepubertal breast, and it is rare before the age of 25. The incidence thereafter increases throughout the entire life span of the individual.

Breast cancer is more frequent in women who have never been pregnant than in those who have borne children. That there may also be a hereditary tendency is suggested by the fact that breast cancer occurs more frequently in the families of breast cancer patients than in the general population.

The cause or causes of breast cancer are unknown. The disturbance which initiates the abnormal growth may be related to hormonal, genetic or viral influences. Benign neoplasms of the breast are probably not a determining factor. Some forms of cystic mastitis (mammary dysplasia) may play a role in the pathogenesis of breast cancer, but this remains an unsettled issue. Local injury to the breast is not regarded as a cause of breast cancer. Frequently, however, the patient will associate an injury to the breast with the discovery of a breast mass. While this circumstance might suggest a causal relationship, such an association is probably coincidental. The injury may simply serve to call the patient's attention to a pre-existing mass.

Breast cancer occurs infrequently in the male, the ratio being about one male to one hundred females. The pathologic features and treatment in the male are similar to those in the female.

In most neoplastic disease, there is a correlation between the duration and size of the tumor, the degree of cellular differentiation and invasiveness and the curability or prognosis. In cancer of the breast, however, these correlations are less well established and can rarely serve as a basis for prognostication. For example, one patient with a large undifferentiated tumor of long duration will remain well indefinitely after radical mastectomy, or will survive many years with known distant metastases; while another with a small, localized, and presumably early, differentiated tumor will die within a few months from distant metastases which appear shortly after operation.

In this regard, MacDonald has called attention to "biologic predeterminism" or the set growth pattern inherent in the tumor from its incipiency. According to this view, some patients with breast cancer are incurable by any form of treatment, some are curable by less than radical operation and some are curable only by radical mastectomy. The maximum theoretical curability rate according to MacDonald's calculations is about 45 per cent of any series, a figure which approximates the over-all salvage rate in this disease.

Most breast cancers arise from ductal epithelium; the remainder, from acinar epithelium. All induce a variable degree of reaction in the supporting fibrous tissue (desmoplasia). Cancers which exhibit marked fibrous tissue reaction are most common and are called *scirrhous carcinoma;* those which contain a paucity of fibrous tissue are called *medullary carcinoma.* The former is hard, fixed and puckered; the latter, rubbery, movable and bulky. Sometimes the cancer cells secrete mucin, in which case the tumor is a *mucinous* or *colloid*

cancer. Paget's disease of the breast is characterized by an eczematoid eruption of the areolae and nipple from infiltrating cancer of the breast. *Inflammatory carcinoma* derives its name from the inflamed appearance of the breast produced by widespread invasion and blockage of lymphatics by cancer cells.

The gross characteristics of scirrhous carcinoma are quite typical. The tumor is irregular, infiltrating and usually woody in consistency.

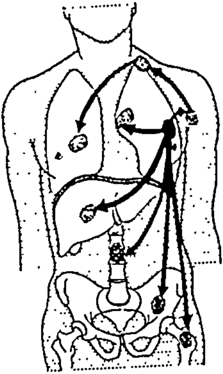

FIG. 121.—Common sites to which cancer of the breast metastasizes: axilla, supraclavicular area, mediastinum, lungs, liver and skeleton. The spread to the skeleton usually involves the spine, pelvis and upper end of the femur, as well as the ribs, shoulder girdle and skull.

When cut, it gives a peculiar gritty sensation, like that of an unripe pear. The cut surface is covered with yellow dots and white streaks. There is no distinct boundary between the tumor and the surrounding normal tissue.

The microscopic picture is variable, but histologic criteria for the diagnosis of a malignant process are demonstrable. There is a remarkable lack of uniformity in the size and shape of the epithelial cells (pleomorphism), as well as a loss of normal cell polarity. An abnormal degree of cell division is indicated by the presence of

numerous mitotic figures. The neoplastic cells also take a deeper stain than do normal cells, or are said to be "hyperchromatic." Finally, there is evidence of invasion of normal tissues.

Breast cancer spreads by continuity and to distant areas by embolization and permeation. Lymphatic spread is largely a matter of embolization through lymph channels to regional nodes and thence

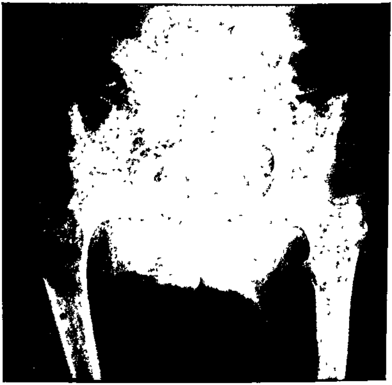

FIG. 122.—Roentgenogram showing metastatic cancer of the pelvis and upper end of both femurs from a carcinoma of the breast Note the "moth-eaten" appearance

to more distant nodes. Occasionally, invading cancer cells enter lymphatics and grow or permeate along the channel, forming cordlike prolongations away from the main tumor. Blood-borne metastases are carried through veins by embolization. Cancer cells may gain entry to veins of the tumor or may enter the blood stream at points where the lymphatic and venous systems unite (e.g., in the supraclavicular region, at the junction of the internal jugular and subclavian veins). Tumor emboli which enter the venous system are widely distributed but appear to survive and grow best in the lung and skeleton (Figs. 121 and 122).

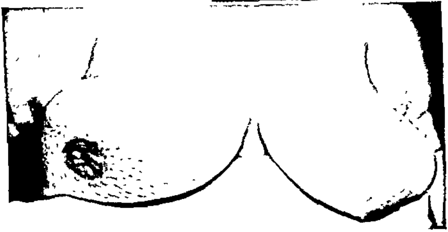

Fig. 123 (*above*).—The retraction sign (dimpling sign) in cancer of the breast. Note also the inversion of the nipple

Fig. 124 (*below*).—"Pigskin" appearance of the breast in advanced mammary cancer.

Clinical Signs.—In addition to a mass, the following signs are pertinent in the diagnosis of breast cancer:

Retraction.—The retraction or dimpling sign (Fig. 123) is the most important single sign of cancer of the breast. It is caused by the contraction or shortening of the fibrous supporting stoma (Cooper's suspensory ligaments) as it becomes involved by the cancer. As the skin is pulled inward, the area is flattened or actually dimpled. If the tumor lies near the nipple, its axis may be altered or the nipple may

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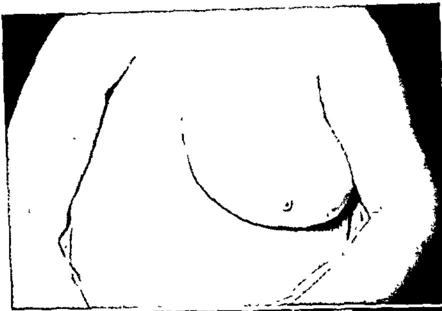
FIG. 122.—Roentgenogram showing metastatic cancer of the pelvis and upper end of both femurs from a carcinoma of the breast. Note the "moth-eaten" appearance.

to more distant nodes. Occasionally, invading cancer cells enter lymphatics and grow or permeate along the channel, forming cordlike prolongations away from the main tumor. Blood-borne metastases are carried through veins by embolization. Cancer cells may gain entry to veins of the tumor or may enter the blood stream at points where the lymphatic and venous systems unite (e.g., in the supraclavicular region, at the junction of the internal jugular and subclavian veins). Tumor emboli which enter the venous system are widely distributed but appear to survive and grow best in the lung and skeleton (Figs. 121 and 122).



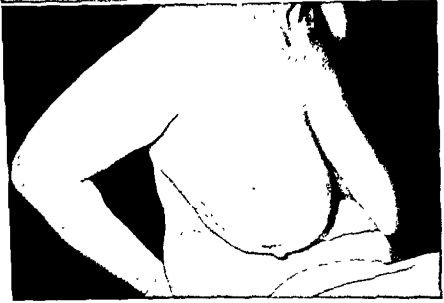


Fig 125 (*above*).—Bloody discharge from the nipple in a patient with cancer of the breast. She previously had had a radical mastectomy on the opposite side for the same disease The second tumor was considered another primary cancer, and not the result of metastases.

Fig 126 (*below*).—Carcinoma of the right breast with extensive skeletal metastases The patient had been treated for chronic backache for several months before the breast tumor was discovered Note the deformity of the skin of the breast, the prominence of a superficial vein and the retraction of the lateral portion of the areolae which appeared when the patient contracted the pectoral muscles.

become inverted. With a change in the axis of the nipple, the nipple is said to "point" in the direction of the tumor.

Edema of the Skin.—The skin over the tumor sometimes resembles "pigskin" (Fig. 124) or "orange peel." This change in appearance is due to blockage of the subdermal lymphatics by cancer cells and to swelling of the skin between fixed points where it is anchored by the skin glands. Edema of the skin is an ominous sign and, if extensive, suggests incurability.

Nipple Discharge.—Infrequently, cancer of the breast causes an abnormal nipple discharge (Fig. 125); and when it does, the discharge is usually blood stained. A discharge is most likely to be due to duct papilloma, cystic disease or plasma cell mastitis.

Inflammation.—Signs of inflammation are unusual in cancer of the breast. The clinical type of cancer known as "inflammatory cancer" is an exception. In this condition the breast is swollen, painful, tender, red and hot. There is no associated suppuration or systemic reaction. The changes are the result of rapid widespread involvement of the breast lymphatics and blood vessels.

Eruption of the Areola and Nipple.—When chronic, this condition indicates the presence of an underlying breast carcinoma until proved otherwise. The superficial breast changes are the result of intra-epithelial and lymphatic spread of the tumor. Oftentimes the skin lesion has a tendency to heal partially, then to recur and progress. A biopsy of the superficial lesion should always be made even though there may be no palpable tumor of the breast.

Satellite Nodules.—Tumor implants in the skin occur in advanced untreated and treated breast cancer. They are often seen in the region of the mastectomy scar months or years after operation. They are firm, fixed masses of variable size located in the skin and subcutaneous tissue, which often undergo necrosis and ulceration.

Fixation of the Breast.—Neoplastic invasion of the fascia, muscles or thoracic wall causes fixation of the breast. In advanced cancer the breast is shrunken (Fig. 126), diffusely hard and immobile. The tumor may extend by infiltration to encircle a wide area of chest wall, giving it an appearance suggestive of body armor, called *cancer en cuirasse* by the French.

Ulceration.—The skin over the tumor may ulcerate as a result of invasion, pressure necrosis or injury. When ulceration occurs, secondary infection follows. The clinical signs of inflammation are then superimposed, and it may be difficult to determine the actual extent

presence of incurable disease. Radical surgical treatment under these circumstances is generally contraindicated because it accomplishes *nothing*—*in* fact, it *is* usually harmful. Patients who exhibit the following signs are classified as "categorically inoperable" by Haagensen and Stout:

1. When the carcinoma develops during pregnancy and lactation (*Note.*—There are rare patients who may be benefited by surgical treatment.)
2. When there is extensive edema of the skin over the breast
3. When satellite nodules are present in the skin over the breast
4. When intercostal or parasternal nodules are present
5. When there is edema of the arm
6. When *proved metastases are found in the supraclavicular nodes*
7. When the carcinoma is of the inflammatory type
8. *When distant metastases are demonstrated in the lungs or bone*
9. When *two* or more of the following signs are present:
   a) Ulceration of the skin
   b) Edema of the skin of limited extent
   c) Solid fixation of the tumor to the chest wall
   d) Axillary nodes measuring 2.5 cm. or more and proved to contain metastases by biopsy
   e) Fixation of the axillary nodes to the skin or deep structures of the axilla

Surgical Treatment.—Excision of breast masses should be undertaken only when all arrangements have been made for immediate pathologic examination of the specimen (frozen section if necessary) and for performing radical mastectomy, if indicated. When this rule is violated, the patient's chances for cure are jeopardized unnecessarily.

The biopsy specimen is obtained after the patient has been placed under general anesthesia. Depending on the size and accessibility of the mass, it may be excised or a small segment may be removed by incision. It is usually advisable to await the pathologist's report before proceeding with radical mastectomy, despite the fact that the diagnosis may be apparent. The report should be available within ten minutes.

If the lesion is benign, simple excision and closure of the wound is often adequate. If the tumor is malignant, the wound is closed and sealed off and the operative field is reprepared. Radical mastectomy is then undertaken, using a fresh surgical setup to guard against the implantation of viable cancer cells from the biopsy wound to the mastectomy wound.

The conventional radical mastectomy embodies the principles of

of the cancer. It is a common error to associate ulceration with far advanced cancer because the clinical signs of cancer and inflammation are quite similar.

Enlarged Axillary Nodes.—Single or multiple, hard and fixed axillary nodes associated with the signs of cancer of the breast warrant a presumptive diagnosis of axillary metastases. However, the absence of palpable axillary nodes does not necessarily mean the absence of metastases. It is well known that in the clinical examination of the axilla grave errors occur in both directions. Clinically positive nodes are found to be histologically negative in about 15 per cent of patients, and clinically negative nodes are histologically positive in 33 per cent of patients. It is apparent, therefore, that certain diagnosis depends on histologic examination of the nodes rather than on simple palpation

Location of the Tumor.—About three fourths of all breast cancers occur in the outer half and central zone, one half in the upper outer quadrant including the axillary tail. Tumors of the outer half of the breast have a better prognosis than tumors of the medial half, presumably because they metastasize to the accessible axillary nodes more often than to the internal mammary nodes.

DIFFERENTIAL DIAGNOSIS.—The diagnosis of breast cancer should be considered when there is a dominant lump, an area of thickening or induration, an erosion of the nipple or areola or a chronic nipple discharge.

The physician must resist the temptation to rely on physical signs alone in the diagnosis of masses in the breast. Even the most experienced clinician will fall into a trap now and then unless he makes it a rule to advise biopsy on the basis of minimal changes in the breast. From all viewpoints, it is better to make a less accurate clinical diagnosis and to resort to early breast biopsy than it is to make a more precise clinical diagnosis which, to the detriment of all concerned, is later proved wrong.

All benign conditions of the breast may at times simulate malignant disease. There are a few common conditions, however, which even the trained pathologist may be unable to differentiate on gross examination alone. These include: the sclerosing adenosis forms of cystic disease, traumatic fat necrosis, plasma cell mastitis and duct papilloma. Conditions of the breast which usually can be recognized on gross examination include: fibroadenoma, the "blue dome cyst" form of cystic disease, sarcoma, lipoma and sebaceous cyst.

Certain signs of advanced breast cancer usually indicate the

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The conventional radical mastectomy embodies the principles of

the Halsted-Meyer operation (1894) and entails the excision, in continuity, of the breast and overlying skin, both pectoral muscles, and the axillary lymph nodes and fat. The long thoracic and thoracodorsal nerves (to the serratus anterior and the latissimus dorsi muscles, respectively) are preserved, if possible, in order to help insure good function of the arm postoperatively. In some instances, skin grafting is necessary for wound closure.

Both the operative and postoperative phases of treatment require more than ordinary attention to many details. Sharp dissection, meticulous hemostasis, rigid aseptic technic, obliteration of dead spaces, avoidance of tissue tension and adequate immobilization of the skin flaps as advocated by Halsted are but a few of the requisites for primary healing and minimal complications.

As soon as feasible, mobilization of the arm with a view to early rehabilitation is begun. Likewise, an artificial breast of proper size and shape should be secured and applied as early as possible. The surgeon should appreciate that the patient has suffered severe emotional, as well as physical, trauma; and in keeping with the dictum to "treat the patient as a whole," he must maintain a helpful and understanding interest.

Two diverse types of surgical treatment have received attention in recent years, but, as yet, neither is widely used. The first consists of simple mastectomy followed by intensive postoperative irradiation (McWhirter of Edinburgh); the second is an extended radical mastectomy with internal mammary and sometimes supraclavicular node dissection (Urban and Wangensteen in the United States, and Margottini in Italy).

The surgical treatment of inoperable breast cancer is limited to biopsy of the tumor and/or its metastases, and simple excision of the breast in selected cases. Sometimes excision of a portion of the chest wall is indicated for localized recurrent cancer.

RADIOTHERAPY.—Radiotherapy is effective in the palliation of breast cancer. Often it is the sole form of local treatment for patients with inoperable cancer. Some radiotherapists advise radiation preoperatively or postoperatively, or both, to selected patients, but there is no unanimity of opinion as to the effectiveness of combined surgical and irradiation treatment. At present, radiotherapy finds its greatest usefulness in the control of metastases, relief of bone pain and healing of pathologic fractures. An important indirect approach to the control of advanced breast cancer entails the inhibition of ovarian

function (specifically estrogen production) by external irradiation.

HORMONE TREATMENT.—Some breast cancers, but not all, are said to be "hormone dependent." The hormone-dependent tumors exhibit changes *in growth activity according to the concentration of certain* hormones in their environment. The male sex hormones (androgens) and the female sex hormones (estrogens) are most influential in altering the growth of breast cancer. Because there are no available methods by which the hormone-dependent tumors can be recognized, management of patients by hormonal alteration involves observing the clinical response to treatment.

All *forms of hormone therapy are* palliative, not curative. About half of the patients treated by these methods derive subjective benefits, and about half of these *will show objective improvement*. The favorable effects of hormone treatment include: relief of pain, feeling of well-being, return of appetite, weight gain, increased strength and vigor, healing of ulcerated areas, regression of the primary tumor and its metastases, healing of pathologic fractures, etc. The improvement lasts for a variable period (usually for several months) if treatment is continued. Then the patient usually begins to lose ground, and the tumor appears *to have escaped from control* of the particular form of hormone therapy in use. Another form of hormonal alteration can then be *tried, and is often effective;* but the favorable response is likely to be of shorter duration than the previous response. In this manner, patients can often be maintained in a relatively comfortable and active condition for a prolonged period.

All attempts to alter hormone balance are attended by some adverse systemic effects. These may be slight and of little consequence, or so severe as to preclude long-term treatment. As with all treatment, the physician must be sure that the end justifies the means.

The androgens are widely used for hormonal alteration and may be given to patients at any age. Their chief benefits lie in their anabolic effect on metabolism and their relief of pain due to bone metastases. They cause *masculinization (deepening of the voice, growth of facial* hair, enlargement of the clitoris, increased libido, etc.) and water and electrolyte *disturbances. Testosterone propionate in a dosage of 100* mg. intramuscularly three times weekly is usually given.

The estrogens are administered only to postmenopausal women. If given earlier, they are likely to accelerate the growth of breast cancer They are most useful in the control of soft-tissue, rather than bone, involvement. Their adverse effects include: nausea and vomit-

ing, vaginal bleeding, water retention and hypercalcemia. Diethylstilbestrol daily in the dosage of 10–15 mg. orally is usually prescribed.

Cortisone and related substances also exert a temporary restraining influence on the growth of breast cancer and often provide a period of subjective improvement. However, the metabolic disturbances which cortisone induces limit its use.

Other methods of hormonal alteration, based on ablation of portions of the endocrine system, are being used or investigated. These include: bilateral oophorectomy, bilateral adrenalectomy and hypophysectomy. The place which these measures will have in the future remains to be determined.

Results of Surgical Treatment.—The operative mortality from radical mastectomy is low, averaging less than 1 per cent. The chief postoperative complications include: wound infection and skin necrosis, edema of the arm, atelectasis and pneumonia, cardiac failure and thromboembolic phenomena.

The five year survival without evidence of disease is the basis of most reports on the results of treatment of breast cancer. It is well known that many of the patients who, at the end of five years, may appear cured are not cured, because metastatic cancer often does not become manifest until after five years or even ten, fifteen or twenty years after radical mastectomy. In addition, the fact that about 20 per cent of the patients with untreated cancer of the breast live for five years or more complicates the interpretation of survival statistics. The patients treated by radical surgery are a selected group and represent only a fraction of all patients with cancer of the breast. The criteria of selection, which varies from clinic to clinic, will also tend to weight the statistics for better or worse, as the case may be. Furthermore, unless the follow-up on every patient is complete, the statistics are inaccurate. For these reasons, it is sufficient that the student have a working knowledge of the five year survival rates for patients with operable breast cancer. The rates are:

The over-all five year survival rate, about 45 per cent

The five year survival rate for patients without axillary metastases, about 75 per cent following radical mastectomy

The five year survival rate for patients with involvement of the axillary nodes, about 25 per cent following radical mastectomy

The foregoing figures are oversimplified for the reader's benefit. The survival rate is reversed according to the status of the axillary lymph nodes on pathologic examination; that is, after five years, if the

nodes are negative, three patients are well and one has evidence of cancer or is dead; if the nodes are positive, one patient is well and three have evidence of cancer or are dead.

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CHAPTER 28

Peripheral Vascular Diseases

An ischemic limb resembles the inhabitants of a beleaguered northern town. With supplies diminished or cut-off, the inhabitants can keep up normal appearances for a time, then they begin to starve, they feel cold, their faces become pale or blue, they are less active, and as conditions grow worse they either become apathetic or complain loudly to those in authority. In the human limb there is absence of arterial pulsation, lowering of surface temperature, pallor or cyanosis, anesthesia and pain.—BLACKWOOD.*

THE PERIPHERAL vascular diseases include those conditions of both structural and functional origin which influence the systems of fluid exchange to the extremities. Included are vaso-obliterative and vasospastic diseases involving the arterial, venous and lymphatic channels, singly or in combination. Because structure and function are interdependent and the systems of fluid flow are influenced by similar mechanisms, disturbances in one system usually also affect other systems. The various conditions encountered can be classified as follows:

Arterial Disease

1. Vaso-obliterative: arteriosclerosis, often associated with diabetes mellitus, thromboangiitis obliterans (Buerger's disease); thrombosis and embolism, aneurysm and arteriovenous aneurysm
2. Vasospastic: Raynaud's disease, acrocyanosis, cold sensitivity and possibly causalgia

Venous Disease

1. Varicose veins
2. Venous thrombosis, thrombophlebitis, phlebothrombosis

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and impulses are mediated through the thoracolumbar chain to both the upper and lower extremities.

Peripheral vasoconstriction may be produced in the following ways:

1. Reflex vasoconstriction (response to cold)
2. Vascular injury or disease
3. Adrenomimetic or related compounds (epinephrine, nor-epinephrine, Neo-synephrine,® ergotamine tartrate)

Peripheral vasodilatation may be effected in the following ways:

1. Vasodilator drugs (aspirin, alcohol, nitrates, tetraethyl ammonium compounds, Priscoline,® Mecholyl,® nicotinic acid, etc.)
2. Reflex vasodilatation (by immersing the hand in warm water; localized vasodilatation also produces generalized vasodilatation)
3. Venous congestion (Buerger's exercises; intermittent venous occlusion; oscillating bed)
4. Sympathetic nerve interruption (direct blockade—procaine or sympathectomy)

Some of the items of importance in assessing blood flow in the peripheral vessels follow:

Pain.—Severe in acute occlusion, pain may be minimal or marked in chronic occlusion, and often nocturnal. With nerve involvement, pain may be unbearable, or sometimes absent in spite of advanced ischemic changes. *Intermittent claudication,* or effort claudication, is a cramplike pain, occurring most commonly in the calf on walking. It disappears on resting, only to recur when walking is resumed. It may be considered "the cry of the ischemic muscle."

Color and Temperature.—Other things being equal, changes in color and temperature reflect changes in blood flow in the affected extremity. So far as possible, the opposite extremity should be used as the control in such observations. Changes in color which occur in response to changes in position (pallor with elevation, and rubor, reticular lividity or cyanosis with dependency) are indicative of ischemia.

Changes in the Skin and Its Appendages.—Atrophy, ulceration, gangrene, brittleness and loss of hair or changes in the nails, edema and pigmentation may occur with changes in peripheral blood flow.

Absence of Pulse.—The pulses in the foot may be imperceptible in normal individuals. However, absence of the pulse in the diseased extremity and presence of it in the opposite member is usually a

3 Postphlebitic syndrome (stasis syndrome)

Lymphatic Disease

1. Lymphangitis and lymphadenitis
2. Lymphedema
   - Congenital lymphedema (Milroy's disease)
   - Obstructive lymphedema
   - Elephantiasis

ARTERIAL DISEASE

The result of either organic or functional diminution of the peripheral vessels is reduced blood flow and ischemia. The effects of ischemia, in turn, are both functional and structural. Thus the ability to initiate and sustain active movement is impaired. There may be intermittent claudication or resting-limb pain; and hyperesthesia, or a "stocking" type of anesthesia. Degenerative changes affect all of the tissues of the extremity; and there may be atrophy, nerve degeneration, necrosis and gangrene.

The factors which modify the effects of ischemia are: the available blood supply, the rate at which the ischemia develops and its duration, and the metabolic demands of the tissues.

The available blood supply is determined by the degree of obliteration of the arterial lumen, the site at which the obstruction occurs and the degree of collateral circulation which develops. Vasospastic and organic peripheral vascular diseases are often associated. Other things being equal, the part which vasospasm plays is inversely related to the age of the patient. *Obstruction at several different levels is more damaging than arterial occlusion at a single area, where collateral channels may be extensive.* Collateral circulation forms best around the joints. *Vasospasm may render this accessory circulation useless.*

Rapidly developing ischemia is less well tolerated than slowly developing arterial insufficiency.

The metabolic demands of the tissues and the changes due to ischemia are increased by heat, inflammation and muscular exercise. The avoidance of local heat, infection and unusual exercise therefore minimizes the adverse effects of ischemia.

The arterioles are under the control of the autonomic nervous system. At least two sets of nerves—the vasoconstrictors and the vasodilators—maintain a normal state of balance in the smooth muscle of the arteriolar wall. The vasomotor nerves are under the control of *vasomotor mechanisms in the medulla, diencephalon* and forebrain;

ances. As such, they may be a source of diagnostic difficulty. For example, thrombotic occlusion of the terminal aorta produces the Leriche syndrome, which may simulate herniated intervertebral disk. There is pain in the back, buttocks, thighs and legs with exercise. Fatigue, weakness and atrophy of the muscles are observed. Impotence in the male is common. The arterial pulses are diminished or absent below the bifurcation. Coldness and pallor of the feet and legs are noted. Gangrene of the toes or foot may appear later. The obstruction can be demonstrated by aortography (Fig. 131, p. 594).

Although vasospasm is generally not a prominent associated feature in arteriosclerosis obliterans, all measures directed toward the relief of even minimal degrees of spastic obstruction are worth while. Thus the physician may provide relief of pain and a measure of "borrowed time," so far as the life of the limb is concerned. This is the rationale of giving vasolytic drugs and performing lumbar sympathectomy in selected cases of arteriosclerotic peripheral vascular disease.

THROMBOANGIITIS OBLITERANS.—Thromboangiitis obliterans, or Buerger's disease, is a chronic inflammatory disease producing obliterative changes in arteries, veins and lymphatics, chiefly of the extremities. The disease, of unknown etiology, occurs predominantly in young or middle-aged men who are heavy smokers. Intermittent claudication and resting-limb pain, and cold, moist, cyanotic feet with ulcerations and gangrene of the toes, are common findings. The pedal pulse is absent or diminished. An important sign is a migratory type of phlebitis manifested by tender areas which appear irregularly in the superficial veins throughout the body. Slow progression of the disease is usual, but the course may be self-arrested or may advance rapidly. Vasospasm, particularly during the early phases, aggravates the ischemia of the extremities.

THROMBOSIS AND EMBOLISM.—Sudden occlusions of major peripheral arteries are serious vascular accidents. The changes resulting from thrombosis and embolism are quite similar, and differentiation may be difficult. Basically the changes result in acute ischemia of the limb. The development of gangrene (Fig. 127) depends on the adequacy of the collateral circulation; and the adequacy of the collateral circulation, in turn, is conditioned by the degree of functional and/or organic obstruction. Thrombosis is the result of vascular wall injury of inflammatory, degenerative or traumatic origin. Peripheral arterial embolism is usually secondary to intracardiac thrombosis, resulting from auricular fibrillation or cardiac infarction or from dislodgment

significant observation. If the pulse is absent in the vessels of the foot, it should be sought for in the popliteal and femoral arteries.

Dilated Tortuous Veins; Varicosities.—These conditions also reflect changes in blood flow.

In addition, certain adjuncts to the study of these problems are available, but they are not usually needed to establish a working diagnosis. The following studies may prove useful:

Temperature Studies.—Vasodilatation is produced (reflexly or by pharmacologic block) and the increased blood flow (as indicated by a temperature rise in the part, preferably in a constant-temperature room) is determined by use of a thermocouple.

Oscillometry.—The volume of the pulse at different levels in the extremity is measured with an oscillometer.

Aortography and Arteriography; Venography.—Radiographic observations are made after intravascular injections of radiopaque iodine-containing solutions.

Blood Flow Studies—Isotope studies, plethysmography, and blood-gas analysis are methods which may be utilized under special conditions.

Vaso-obliterative Arterial Disease

Arteriosclerosis (Endarteritis) Obliterans.—This disease occurs in the older age group, most often in men. It may be considered a manifestation of "the wearing-out process." Arteriosclerosis involves both lower extremities as well as other vessels throughout the body. Diabetes mellitus is often associated. Intermittent claudication and resting-limb pain are common symptoms. The feet are usually cold, elevation causes pallor, while dependency causes a slow return of color, which progresses to cyanosis; skin changes may vary from skin atrophy to gangrene; the pulses in the foot, and often in the popliteal and femoral areas, are markedly decreased or absent. The condition tends to be progressive over a long period.

The localized or segmental form of arterial occlusion associated with arteriosclerosis (atherosclerosis) is often amenable to operative correction by removal of the obstructing thrombus with the intima or excision and vessel replacement. The chief sites in which localized occlusion occurs include: aortic bifurcation, iliac, femoral and popliteal arteries. These conditions cause symptoms of vascular insufficiency which may suggest primary neurologic or musculoskeletal disturb-

time, arterial embolectomy is of value only in large vessels soon after embolism has occurred.

Aneurysms.—Blood-filled sacs which communicate with arteries are called "aneurysms." In the extremities, such sacs occur chiefly as a result of trauma (traumatic, or false, aneurysm; pulsating hematoma) or of degenerative disease (arteriosclerosis, syphilis). False aneurysms are formed from hematomas which remain in communication with the lumen of the artery of origin. They are lined, not with endothelium, but by the inner surface of the clot. The original wound is often insignificant, but hemorrhage from it may be severe and out of proportion to what would ordinarily be anticipated. A mass forms and gradually increases in size, producing varying degrees of obstruction to blood flow in the main and adjacent vessels. True aneurysms are most often located in the aorta, iliac femoral or popliteal arteries and may produce serious circulatory deficiency.

The essential physical sign in aneurysm is an expansile mass located over the course of a major artery. The force of the pulsation and other physical signs depend on the size of the communication with the vessel of origin and the degree of clotting within the mass. With proximal occlusion of the artery, the pulsation disappears; with release, it returns. A thrill and bruit may be present, depending on the size of the opening into the aneurysm. The degree of ischemia of the distal tissues will depend on the degree of vascular impairment produced by the mass.

Ideally, aneurysms are treated (Fig. 128) by excision and re-establishment of the continuity of the artery. This may be accomplished by removal of the aneurysm, followed by end-to-end anastomosis or bridging of the gap with a vessel graft. If this type of removal is impractical, ligation of the vessel and removal or obliteration of the aneurysm later may be necessary. Under the latter conditions the survival and function of the limb will depend on the critical factor of collateral circulation.

Arteriovenous Aneurysm or Fistula.—Abnormal communications between large arteries and veins may result from penetrating wounds (traumatic arteriovenous aneurysm) or congenital vascular shunts. The congenital type most often occurs in the scalp (cirsoid aneurysm), but similar lesions are seen in the extremities. The traumatic type usually is produced by gunshot or stab wounds.

During its formation, a traumatic arteriovenous aneurysm resembles a pulsating hematoma; but as the opening between the artery

of thrombotic calcified plaques from the aorta. Progressive thrombosis of the involved vessel usually follows both embolism and thrombosis. The unattached clot of embolic occlusion becomes fixed to the vessel wall (intima) within eight to ten hours. Permanent restoration of continuity of the vessel by embolectomy can therefore be expected only if the embolus is removed during this period of "golden opportunity." If the clot is removed later, existing intimal damage is likely to lead to a secondary thrombosis.

Embolectomy is usually reserved for embolism of the large arterial

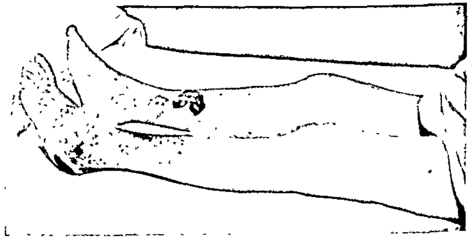


FIG. 127.—Bilateral "dry" gangrene of the legs resulting from a "saddle" embolus obstructing the terminal aorta. The embolus originated from a thrombus in the right auricular appendage. Amputations required: above knee on the right; below knee on the left. Patient recovered. Early embolectomy might have prevented loss of the legs

trunks (iliac and femoral), including the bifurcation of the aorta (saddle embolus) and, occasionally, the axillary artery. In other vessels the collateral circulation may be adequate to support the distal tissues if vasospasm is overcome. Furthermore, the small caliber of the other vessels makes it unlikely that they will remain open after removal of the clot. Narrowing of the lumen and trauma resulting from operation, together with the resultant decreased volume flow, enhances the tendency to recurrent clot formation. Operations for the removal of clots following acute arterial thrombosis are not likely to succeed because intimal damage usually leads to recurrent thrombosis. It is possible that future extensions of the use of anticoagulant measures may allow a more direct approach to this problem. At the present

and the vein is formed, the signs become more clear-cut. There is a gradual relentless dilatation of both the arterial and the venous channels of the regional circulation, with formation of a pulsatile mass containing varicosities under high pressure. There is a palpable systolic thrill and an audible continuous machinery-like murmur. Occlusion of the fistula by compression causes increased resistance to peripheral flow and results in an immediate reflex slowing of the pulse rate (Branham's sign). There are signs of diminished blood flow and ischemia of the extremity, depending on the size of the shunt. Signs of arterial insufficiency are usually progressive. Great enlargement of the extremity may occur, particularly if the fistula is congenital or develops during the growth period. The possibility of an arteriovenous fistula must always be considered when large varicosities which are confined to one extremity or localized to one area of the trunk or head are found. It is obvious that, under these conditions, treatment of the varicose veins alone would be fruitless. Heart failure ultimately results from untreated arteriovenous fistula. Therefore, surgical closure of arteriovenous fistulas is indicated. In the early stages, however, it is sometimes advisable to defer operation for two or three months, in order to allow maximal development of the collateral circulation.

The pathophysiologic changes resulting from the "parasitic circulation" of an arteriovenous fistula are numerous and profound:

1. Initially, the systemic blood pressure is reduced as a result of the decreased peripheral resistance.
2. Later, the systolic pressure rises to normal and the diastolic pressure remains low, since there is a continued decreased resistance. The pulse pressure is therefore high.
3. The pulse rate is increased (Bainbridge reflex).
4. Blood volume is increased.
5. Cardiac output is increased. There is hypertrophy and dilatation of the heart, and later cardiac failure.
6 Closure of the fistula and restoration of blood flow to the normal capillary bed of the extremity reverses the process. The pulse rate slows; the blood pressure becomes stabilized; the cardiac output falls; and the blood volume decreases.

The ideal treatment of the arteriovenous fistula consists in closure of the fistula and preservation of both the main arterial and venous channels. This is sometimes possible without the use of vascular grafts. The time-honored treatment of arteriovenous fistula has been excision and "quadruple ligation" of both the artery and the vein, on either

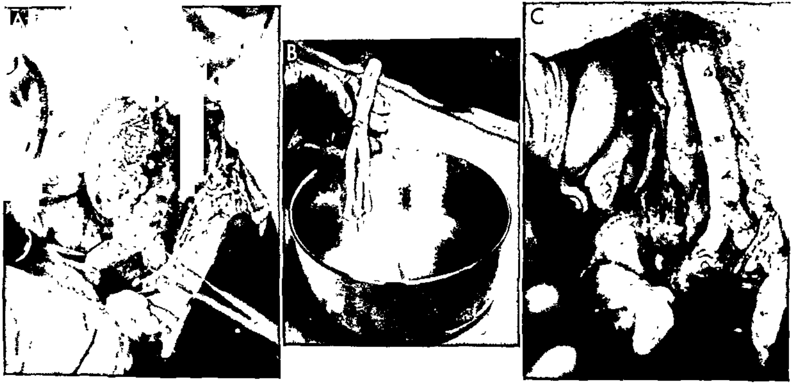


Fig. 128.—Atherosclerotic aneurysm of the terminal aorta treated by excision and replacement with an arterial homograft. *A*, the aneurysm. The renal arteries are not visible but come off the aorta above the aneurysm. The common iliac arteries can be seen at the lower margin of the aneurysm. *B*, the homograft, which was obtained at necropsy and stored after freeze-drying. Normal pliability of the graft is restored by soaking it in saline. The graft must be tailored to fit the defect. *C*, the aneurysm removed and the graft inserted. Note the suture lines which unite the graft with the vessels of the host. Blood is now flowing through the graft. (Courtesy of R. E. Taber.)

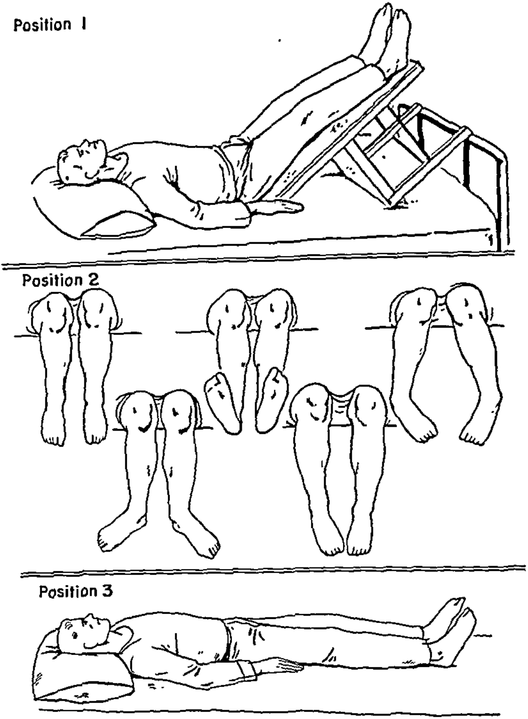


FIG. 129.—Buerger-Allen active vascular exercises. Position 1, two minutes; position 2, three minutes, position 3, five minutes. Repeat the exercises three or four times each exercise period, several times (three or four) daily. (Courtesy of W. D. Paul, State University of Iowa)

side of the fistula. This cures the arteriovenous fistula but usually results in vascular insufficiency of the extremity, and is, therefore, less than optimal treatment.

Vasospastic Arterial Disease

Raynaud's Disease.—Raynaud's disease is a type of intermittent angiospasm which occurs almost exclusively in females; it involves the upper extremities in a bilateral and symmetrical manner. Attacks may be precipitated by exposure to cold or by emotional disturbances. Typically, there are three phases to the attack:

1. Local syncope (the fingers are white)
2. Local asphyxia (the fingers are blue, swollen and painful; pain may be severe)
3. Recovery (the fingers are red, warm or hot; pain decreases; and paresthesias appear)

Atrophy of the skin, ulceration and gangrene of the fingertips and deformities of the nails are the usual sequelae in advanced disease. Raynaud's disease follows a comparatively benign course compared with other major types of peripheral vascular disease. As time goes by, the attacks may become less frequent or disappear entirely, but generally, organic occlusive changes eventually take place. Protection of the hands from cold and injury is of great importance. Sympathectomy in the upper dorsal segments appears to contribute little to the relief of symptoms or to the arrest of the disease. Nonoperative measures, such as protection of the hands from exposure to cold, avoidance of emotional tensions and the administration of vasolytic drugs, probably provide better control than surgical procedures.

Raynaud's Phenomenon (Syndrome).—Symptoms and color changes similar to those described above may also occur in certain other types of occlusive vascular disease (e.g., Buerger's disease), pneumatic hammer disease, heavy-metal or ergot poisoning, cervical rib and some neurogenic disturbances.

Other Vasospastic Diseases.—Less common and little-understood functional conditions include: acrocyanosis, erythromelalgia, cold sensitivity, causalgia and reflex dystrophies.

Treatment in Peripheral Arterial Diseases

Therapy is based on an understanding of the pathophysiologic alterations which these conditions produce, the methods of examina-

6. The local care of the extremities is an important factor (Table 23). The patient should be carefully instructed in all details, verbally as well as in writing, and examined at regular intervals. The feet should be washed daily with soap and warm water, dried carefully, then massaged gently with alcohol or lanolin. Socks should be

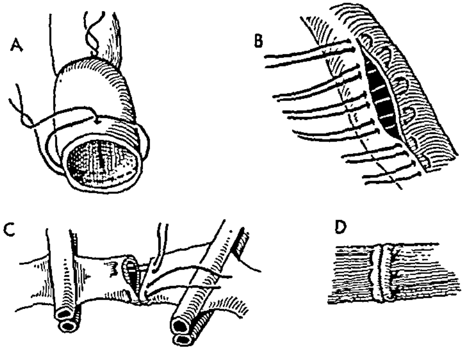


Fig. 130.—Methods of blood vessel closure and anastomosis. (Currently more emphasis is placed on the need for an adequate lumen than on eversion of the intima to prevent thrombosis after blood vessel suture. Simple continuous over and over silk sutures may be used.) *A*, double ligation of large vessels by a proximal encircling ligature and a distal transfixion ligature in order to insure against slippage and hemorrhage. *B*, closure of a rent with interrupted everting mattress sutures (or a continuous, everting suture) of silk in order to leave a smooth internal intimal lining and prevent thrombosis. The lumen must not be narrowed unduly lest thrombosis occur from slowing of blood flow. *C*, end-to-end anastomosis of vessel by dividing the circumference of the lumen into three equal parts with everting mattress sutures. *D*, the circumferential suture completed.

changed daily. Shoes should be well fitted and comfortable. The breaking-in of new shoes should progress slowly. The patient should be instructed to cut nails straight across. He should not cut corns or calluses, but should soften them by soaking in warm water and then rub them off with gauze. Minor injuries or infections of the foot should be a cause for concern, and the patient should not attempt self-treatment. Exercise should be within pain limits.

tion and interpretation, and an appreciation of the intrinsic and environmental factors which influence the course of the disease. One must realize that vascular disease produces widespread changes in the peripheral tissues and that it frequently leads to disturbances in all systems of fluid flow. Also, vaso-occlusive and vasospastic impairment of flow are often coexistent and operate in a vicious cycle. Without special methods of investigation, it may be impossible to assess accurately the role of all factors in the production of the presenting dis-

TABLE 23.—"Do Nots" for Patients with Peripheral Vascular Disease and/or Diabetes (After Wilder)

Do not use commercial corn remedies.
Do not use sharp instruments on corns and calluses.
Do not use iodine.
Do not walk on bare feet.
Do not open blisters with pins or needles.
Do not place hot-water bottles near the feet.
Do not wear circular garters
Do not bathe feet in too hot water.
Do not wear tight or badly fitted shoes.
Do not wear metal arch supporters.
Do not use Epsom-salt foot baths without physician's advice.
Do not apply adhesive tape directly to the skin.
Do not bandage feet tightly
Do not seek your neighbor's advice—consult a physician.

order. The essential points in treatment are: (1) the elimination of factors which produce vasospasm, (2) the utilization of aids to produce vasodilatation, (3) special care of the ischemic parts and (4) removal of the obstruction, if possible.

In treating peripheral arterial disease, the following methods should be employed:

1. Cold, tobacco, emotional disturbances and other adverse influences which enhance vasospasm should be avoided.

2. Buerger-Allen's exercises (Fig. 129) are an effective physiologic means of securing vasodilatation and are said to aid development of collateral circulation.

3. Reflex vasodilatation, by the use of heat (115° F.) applied to the hand or some other portion of the body for twenty minutes three times daily, may be helpful.

4. Vasodilator drugs, such as aspirin, alcohol, Priscoline® and papaverine, are commonly used.

5. Sympathetic interruption—chemical (procaine or similar agents) or surgical excision (for the lower extremities, sympathetic ganglia L2, 3, 4)—may be useful in selected cases.

7. Restoration of arterial flow (Fig. 130) through obstructed segments between the abdominal aorta and the popliteal artery may be accomplished either by resection and replacement grafts or by removal of the clot together with the lining of the vessel (thromboíntimectomy). Arteriography (Fig. 131), especially lumbar aortography, is essential in selecting patients for these procedures. Arterial

Fig. 132.—Refrigeration of the extremity in advancing diabetic gangrene. The patient was a women of 56 with marked generalized arteriosclerosis, cardiac disease, systemic toxicity (fever, high pulse rate, leukocytosis), disturbed sensorium and uncontrollable diabetes. The lower leg was packed in ice for twenty-four hours, after which her general condition improved greatly and an above-knee amputation was performed without complications.

homografts preserved by freeze-drying have proved very satisfactory for this purpose. Plastic fabric prostheses made from nylon, orlon, dacron, etc., have received extensive trial and would appear to act as satisfactory blood conduits. Only a minority of patients with obliterative arterial disease are suitable candidates for these procedures; but when properly selected, the results have proved highly satisfactory.

The treatment of the complications of ischemia, such as ulceration, infection and gangrene, must follow well-established principles. This involves attention to both the local and the systemic factors which influence the response to infection and healing. Rest, clean surgical

ness of the legs and impotence (Leriche's syndrome). At operation the thrombosed segment was removed and replaced with a freeze-dried homograft of the aortic bifurcation. Recovery was uneventful, and the patient was relieved of symptoms.

VENOUS DISEASE

Varicose Veins

It is the impairment of functional efficiency of the tissues secondary to increased interstitial fluid which seems to us at the present time to offer the best physiologic explanation for the heaviness, edema, easy fatigability, and increased susceptibility to infection and trauma which are so distressing to the patient with severe varicose veins.—Barrow.

Abnormally dilated, tortuous and elongated veins are said to be "varicosed." While the term is ordinarily used in reference to varicose veins of the lower extremities, comparable conditions occur in the esophagus (esophageal varices), in the rectum (hemorrhoids) and in the spermatic cord (varicocele).

The venous circulation of the lower extremity is composed of two intercommunicating systems: (1) the superficial system, which consists of the greater (long, internal) saphenous vein and its tributaries and the lesser (short, external) saphenous vein and its tributaries, and (2) the deep system, which consists of the femoral vein and its tributaries.

The superficial system of the veins of the leg is a primary site for the development of varicosities. The superficial veins lie in the subcutaneous tissues and are not protected or supported by muscular and fascial structures, such as those which enclose the deep veins. The greater saphenous vein arises at the dorsal venous arch of the foot and passes upward just anterior to the internal malleolus of the ankle, then medial to the internal condyle of the femur and along the medio-anterior aspect of the thigh to the fossa ovalis. Here it penetrates the fascial opening and joins the femoral vein. The lesser saphenous vein arises behind the external malleolus, passes upward over the posterior surface of the leg and penetrates the fascia over the popliteal fossae to join the deep popliteal vein. Numerous communicating veins ("perforators") exist between the main trunks of the greater and lesser saphenous veins and the deep system of veins of the leg.

The greater saphenous vein joins the femoral vein at the fossa ovalis, where an enlargement, called the saphenous "bulb," is found. Several superficial veins converge at this point to join the bulb. The number and distribution of these vessels is variable, but usually there are at least three veins. These are: (1) the superficial inferior epigastric, (2) the superficial iliac circumflex and (3) the superficial internal

care, adequate drainage, removal of nonviable tissue and the administration of antibiotics constitute the most important items. The prolonged use of heat in any form to ischemic tissues is absolutely contraindicated because it accelerates the metabolic demands of the tissues. The demand cannot be met by the impaired circulation, and gangrene may be precipitated or accelerated. Cooling decreases the metabolic demands of tissue, and it is sometimes of value in the treatment of impending or advancing gangrene. Refrigeration (Fig. 132) or freezing of the parts should be utilized only when the situation is critical—for example, when the foot or leg is obviously gangrenous and beyond salvage. Sometimes in seriously ill patients, such as those with multiple injuries and associated peripheral gangrene of vascular origin (not gas gangrene), or in patients with severe diabetic acidosis complicated by gangrene, operation can be delayed by refrigerating the limb while the more pressing problems of cardiovascular, pulmonary and metabolic origin are corrected. Amputation can then be carried out later under more nearly optimal conditions. From a practical viewpoint, refrigeration of the limb may be considered a physiologic amputation.

The general care of the patient is always important. Diabetes mellitus with peripheral vascular disease makes a more difficult therapeutic problem. Diabetes not only accentuates arteriosclerosis and ischemia, but it also impairs the body defenses and healing potential. The abnormal carbohydrate metabolism of the diabetic patient appears to favor bacterial growth and lowers resistance to invasive infection.

Dry gangrene is due to acute or chronic arterial occlusion, and the veins often are not obstructed. The part becomes cold, black and shriveled. Wet gangrene is the result of arterial and/or venous occlusion, stasis, edema and infection. The part is swollen, cold, ulcerated and discolored. Gradations between these categories are common. In general, wet gangrene constitutes an urgent indication for amputation because of the profound systemic effects resulting from absorption of the products of infection and tissue breakdown, as well as the tendency for the gangrene to spread. Amputation for dry gangrene is sometimes deferred until a line of demarcation develops between the viable and the nonviable tissue. According to the circumstances, excision of a portion of a toe, a foot, a lower leg or a thigh may then be required

valves just below the entrance of tributaries and near the mouths of tributaries. Valves of great clinical importance are located in the communicating (perforator) veins of the leg and in the saphenous bulb. When the valves become functionless or incompetent, the superficial veins are no longer protected from the unphysiologic pressures resulting from straining, standing, etc., and reverse flow occurs.

The factors responsible for the flow of blood in the veins are:

1. The momentum imparted to the blood by the heart and transmitted through the capillary bed (vis a tergo)
2. Muscular contractions aided by venous valves
3. Changes in intrathoracic and intra-abdominal pressure with respiration
4. Possibly the intermittent pressure changes produced by arterial pulsation

Factors which tend to retard the flow of blood in the veins (Fig. 133) are:

1. The hydrostatic pressure or gravity effect of the upright position
2. Increased intra-abdominal or intrathoracic pressure
3. Increased resistance to flow such as that produced by venous obstruction, encircling bandages, garters, etc.
4. Immobilization

Normally, the venous valves allow blood to flow in one direction only (Fig. 134, *A*). Pressure applied to the vein as a result of muscle contraction causes the vein to collapse partially and the blood to be displaced centrally through the opened leaflets of the valve. When the pressure is released as the muscle relaxes, the valve leaflet passively closes and prevents reflux of blood. Muscle contractions acting in conjunction with competent venous valves constitute a "peripheral venous heart" for movement of the blood from the extremities back to the heart

The flow of blood is directed normally from the superficial veins to the deep veins. When abnormal stresses or disease renders the venous valves incompetent (Fig 134, *B*, *C*), there is reverse flow, and a progressively greater load is placed on the superficial veins, leading to the development of varicosities. There then is stagnation of blood and a local increase in venous pressure, which leads to alterations in fluid exchange in all compartments in the leg (vascular, interstitial and intracellular). When there is sustained increased venous pressure, increased leakage of protein-rich fluid from the capillary bed and decreased absorption from the tissue spaces occur. The resulting

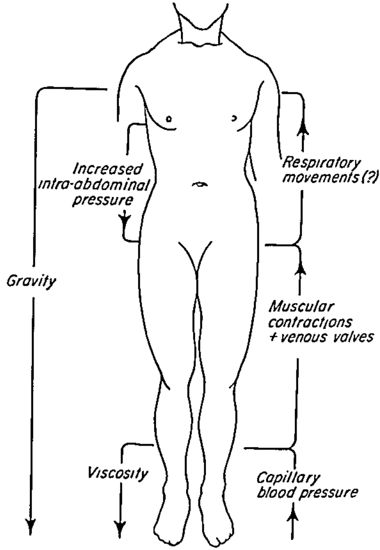


FIG. 133 —Factors normally favoring and opposing venous flow in the extremities. (After Barrow, D. W: *The Clinical Management of Varicose Veins* [New York: Paul B Hoeber, Inc, 1948])

pudendal. These tributaries are important collateral channels and may be responsible for recurrent saphenous varicosities after operation, unless each vessel is individually ligated and divided at the primary operation for varicose veins.

Venous valves are reduplications of the intima, which (in conjunction with muscular contractions) help to move blood in the direction of the heart and to prevent backward or retrograde flow of blood in veins. The superficial and the deep veins of the extremities contain

toward the end of the day. Frequently, the varicosities become more prominent, and aching or pain and tenderness are increased, during the menstrual period. As the condition progresses, edema may become constant and fibrosis with induration of the subcutaneous tissues develops. Pigmentation of the skin, usually above the medial side of the ankle, and stasis dermatitis or eczema may appear. Chronic ulceration, especially in the region of the medial malleolus, is prone to follow slight trauma and infection. The healing response is impaired, and often the ulcer will be resistant to all local treatment. Chronic infection in the ulcer may spread and lead to thrombophlebitis, lymphangitis or cellulitis. Enlargement of the varicosities may follow increased venous obstruction when thrombophlebitis is superimposed. Rupture of a varix with hemorrhage sometimes occurs spontaneously or is produced by slight trauma.

EXAMINATION.—The investigation cannot be confined to the lower extremities alone. It must include a complete history and physical examination. Because intra-abdominal, intrathoracic or primary vascular conditions (e.g., arteriovenous aneurysm) may lead to the formation of varicose veins, these possibilities should be considered. A history of previous thrombophlebitis of the deep veins or of milk-leg (phlegmasia alba dolens) will indicate the need for special care in evaluating the disturbance and prescribing any type of treatment. The laboratory examination is directed toward determining the presence of underlying or associated diseases, such as heart disease, syphilis and diabetes mellitus.

Following the general examination, attention is directed toward the evaluation of the varicosities (Fig. 135). Certain questions must be answered:

1. Are the varicosities located in the greater or lesser saphenous system or both?
2. Is the valve at the saphenous bulb incompetent?
3. Are the valves in the communicating veins (perforators) incompetent?
4. Where are the incompetent perforators located?
5. Is the deep venous system blocked by previous thrombosis?

Each patient presents a different problem, and treatment must be individualized according to the circumstances. Several clinical tests (Trendelenburg, Perthes and Mahorner-Ochsner) are utilized in order to determine the location and degree of disturbed valve function.

Trendelenburg Test.—1. Positive Constriction Test (also called

subclinical or overt edema with chronic anoxia leads to fibroblastic proliferation and induration of the lower leg. The lymphatic channels are overloaded and may become partially obliterated or thrombosed and incompetent. The osmotic pressure of interstitial fluid locally is relatively increased and further tends to embarrass the circulation. Ultimately, permanent alterations (swelling, induration, pigmentation, ulceration, eczema, etc.) appear. If permanent changes in the leg are to be prevented, venous insufficiency must be recognized and treated while it is still reversible.

The etiologic factors in the development of varicose veins are: (1)

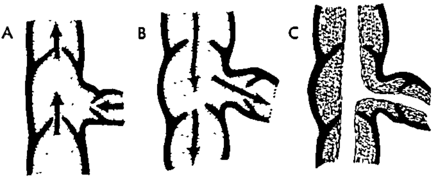


FIG 134 —The function of venous valves as influenced by (1) dilatation of the vein wall and (2) thrombosis and recanalization of the vein lumen. *A*, normal competen [illegible] of the vein. [illegible] This [illegible] el has been [illegible] This is often [illegible]

congenital weakness of the vessel wall and valves, or absence of valves, and (2) activity stresses and other causes which markedly increase intra-abdominal pressure and venous pressure.

It is necessary to distinguish between increased venous pressure due to simple gravity effect and the activity stresses and increased venous pressure due to pathologic conditions. Among the latter are: ascites, pregnancy, abdominal tumors, portal vein obstruction, deep venous occlusion and congenital or acquired arteriovenous communications.

SYMPTOMS AND SIGNS —Except for their unsightly appearance, varicose veins may give rise to no complaints. More often, however, there is heaviness and easy fatigue of the legs on standing, aching pains in the calves and swelling about the ankle, the latter most noticeable

"Trendelenburg Negative").—With the patient supine, the leg is elevated, emptying the veins. A tourniquet is applied at the thigh, high enough and tight enough to block the flow in the superficial veins. The patient stands upright, and the time that is required for the varicosities to fill is noted. If the filling takes thirty seconds or longer, it is

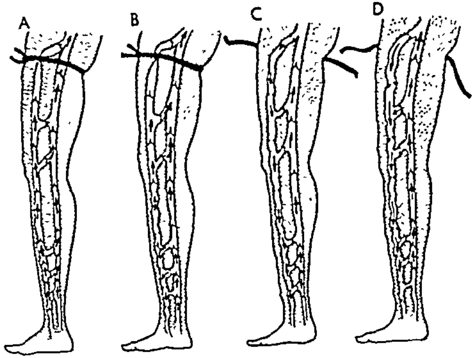


FIG. 136.—The Trendelenberg test for varicose veins (see text for explanation). (Immediately before having the patient stand, the veins are emptied by elevating the leg and then occluded by applying a tourniquet to the thigh below the fossa ovalis) *A*, normal or negative constriction test. Note the normal direction of blood flow as indicated by the arrows *B*, positive constriction test. Note the reflux through the communicating veins from the deep to the superficial leg veins. *C*, positive release test. Note the reflux through the saphenofemoral junction from the deep to the superficial veins. *D*, double positive constriction-release test. Note reflux from the deep veins through both the communicating veins and the saphenofemoral junction.

probable that the valves of the communicating veins are normal (competent) (Fig 136, *A*), if less than thirty seconds, the presumption is that these valves are functionless (incompetent) and that the superficial veins have filled by reflux from the deep veins (Fig. 136, *B*).

2. Positive Release Test (Fig. 136, *C*; also called "Trendelenburg Positive").—The leg is again elevated, and a tourniquet is applied at the thigh. The patient stands, and the tourniquet is released. If the varicosities fill immediately from above, the inference is that the valve

FIG 135 —Severe bilateral varicose veins in a male patient. Note the prominent internal saphenous veins in the thighs and the marked varicosities of the terminal branches over the inner surfaces of the lower legs and ankles. Studies revealed incompetence of the valves at the saphenofemoral junction and of the valves in the communicators at the knee region bilaterally. Bilateral saphenous "stripping" was done, and dissection with ligation of the incompetent communicators. The results, on follow-up, were satisfactory.

has no pain or discomfort over a period of several days and can wear the bandage continuously, the deep veins can be considered capable of returning all the blood from the extremity even when the superficial venous system is occluded. If swelling and symptoms (fatigue, aching, etc.) disappear, it is likely that treatment for varicose veins will be effective. On the other hand, if the patient has increased pain and swelling and cannot tolerate the bandage, the inference is that deep venous obstruction exists, and treatment for the varicosities must be applied cautiously, if at all.

Mahorner-Ochsner Test.—This test is a modification of Perthes' test. A tourniquet is applied high on the thigh, and the veins are observed while the patient walks. If the varices empty, only the valve at the saphenous bulb is incompetent. If the varices do not empty, the tourniquet is applied at progressively lower levels and the observations are repeated. The level at which emptying occurs indicates the lowest incompetent perforator.

Other tests (Pratt, Harkins, etc.) for localization of incompetent communicating veins are useful in the detailed study of complicated cases.

TREATMENT.—The therapy of varicose veins has passed through several phases. Originally, surgical excision with "stripping" was practiced in severe cases Later, injection of sclerosing solutions became popular. Then a combination of ligation of the saphenous vein and its tributaries with injections of sclerosing solutions came into vogue In recent years, there has been renewed interest in stripping operations. In these procedures the main channels of the greater or lesser saphenous systems, or both, are surgically removed or stripped. The veins which communicate with the deep venous system are avulsed during stripping and become thrombosed, or they may be individually dissected out and ligated. Stripping operations are at present widely used in the treatment of primary and recurrent varicose veins. Varicosities which persist following stripping may be thrombosed and obliterated by careful injection of sclerosing solutions.

General Rules of Treatment.—The following general rules should be observed:

1. Deep venous thrombosis is usually a contraindication to vein ligation, stripping or injections of sclerosing solutions.

2. If the valves of the saphenous bulb and the communicators are competent (there is no reflux) and only small varicosities exist, no treatment may be required, or consideration may be given to local injections of sclerosing solutions.

at the saphenous bulb is incompetent and abnormal reflux of blood has occurred from the femoral vein into the saphenous system.

3. Double Positive Constriction-Release Test (Fig. 136, *D*).—The first test is repeated. If the varicosities fill in less than thirty seconds with the tourniquet in place and immediately become more distended after release of the tourniquet, this is interpreted to mean that the valves between the deep and the superficial systems and the valve at the saphenous bulb are incompetent. Reflux of blood has occurred from the deep system through functionless valves in these areas.

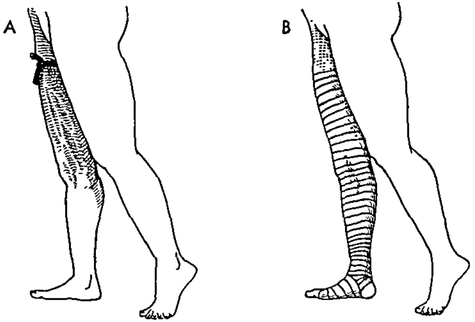


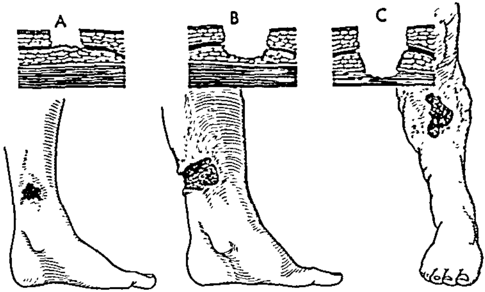
FIG 137.—Tests for patency of the deep venous circulation of the leg. *A*, Perthes' test. *B*, compression test. (See text.)

Perthes' Test.—In making Perthes' test (Fig. 137, *A*), a tourniquet is applied at the thigh and the patient is instructed to walk around for several minutes. If the varicosities become more prominent and painful, there probably is obstruction to flow in the deep veins. This is usually due to a previous deep venous thrombosis. If the communicating veins and valves are functional and the deep system is open and adequate (normal), the superficial varicosities will collapse when the pumping action of muscular contraction forces blood from the superficial veins into the deep system.

Another test (compression, Fig. 137, *B*) for the patency of the deep veins of the leg consists of having the patient wear a circular elastic bandage around the entire leg when ambulatory. If the patient

and anterior to the internal malleolus. The ulcers may be single or multiple, and they have punched-out margins and a granulating base. There is associated induration of the regional subcutaneous tissue, and often a "feeder vein" can be seen leading to the ulcer.

The objectives of treatment are: to improve or restore normal tissue metabolism, to overcome infection, to secure healing of the ulcer and to prevent recurrence. The accepted treatment consists of



[illegible] depth of penetration. [illegible] s may not be marked. [illegible] be deep, irregular and extensive Local skin changes consist of edema, induration and pigmentation. Varicosities may or may not be present. C, arteriosclerotic ulcer. Ulcers are deep, irregular and usually located over the tibial area. Associated changes are due to arterial insufficiency rather than to venous stagnation

the following: elevation of the leg to eliminate the adverse effects of hydrostatic pressure, warm saline packs and antibiotics to overcome infection and aid healing; obliteration or removal of varicosities and incompetent perforators, and utilization of external compression bandages when the patient is ambulatory. It is probable that the type of ointment or local antibiotic used makes little difference in the rate of healing. Operative procedures on the veins are contraindicated during the acute inflammatory phase of ulceration and must be deferred until infection has been controlled. Uniform external compression aids tissue fluid exchange and venous drainage. Such compression may be achieved by applying circular elastic bandages, elastic stock-

3. If the valve at the saphenous bulb is incompetent and the valves of the communicators are competent, high ligation of the saphenous vein (and tributaries) with stripping of the vein is accepted practice. If there is evidence of varicosities in the lesser saphenous vein, this vein should also be stripped.

4. If the valve at the saphenous bulb is incompetent and the valves of the communicators are also incompetent, ligation and stripping of the saphenous vein and dissection and ligation of the communicators should be performed. Multiple incisions are usually necessary. In some instances, injections of sclerosing solutions may be required later.

5. Varicosities of the lesser saphenous system may be treated by ligation of the lesser saphenous vein in the popliteal fossa at its juncture with the popliteal vein and stripping.

Local Injection Treatment.—Injection of sclerosing solutions into varicose veins produces chemical changes in both the vein wall and the blood. A thrombus is formed which ultimately undergoes organization. Canalization of the thrombus and recurrence of varicosities often occur if the thrombosed vein is in communication with open veins. A recanalized vein is a poor substitute for either a normal or a thrombosed vein because the wall of the recanalized vein is thick, its valves are destroyed and stagnation of blood within the vein occurs.

A large number of sclerosing solutions are available Sodium morrhuate (5 per cent) is perhaps the most widely used agent. Because severe or even fatal sensitivity to this drug occasionally occurs, a test dose should be given on the first visit. If there is no evidence of sensitivity, injections are usually given a week apart in divided doses into several areas. The possibility of late sensitivity requires repetition of the test dose when there are long intervals between injections All intravenous injections are potentially dangerous and must be administered with great care. The danger of producing a deep thrombosis from injection of superficial veins must be kept in mind. It is surprising that this serious complication does not occur more frequently than it does.

Complications of Varicose Veins

Varicose Ulcer.—The combined effects of chronic malnutrition of the tissues of the lower leg plus trauma with secondary infection often lead to varicose ulcer (Fig. 138). Usually, the ulcer is located above

ings or an Unna paste boot. In resistant varicose ulcers, skin grafts may be necessary to achieve rapid coverage.

STASIS DERMATITIS.—The pathogenesis of stasis dermatitis (Fig. 139) is similar to that of varicose ulcer. However, varicose veins may be small or absent in this condition. Secondary mixed bacterial or fungus infections commonly complicate the eczema. Treatment of

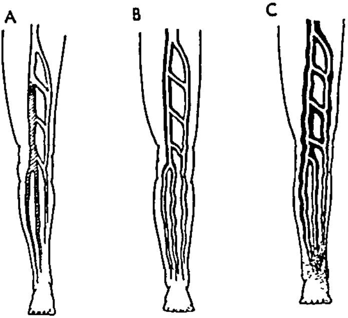


FIG. 141.—Pathophysiology of the postphlebitic syndrome of the leg. *A*, acute thrombotic occlusion of the deep veins with destruction of venous valves. Edema of the leg. *B*, recanalization of thrombosed veins of the deep system, but valves have been permanently destroyed. *C*, varicosities of the superficial veins and chronic edema, induration, pigmentation and ulceration due to unrelieved venous hypertension in the upright position and failure of the "peripheral venous heart."

stasis dermatitis requires correction of the disturbed circulation and elimination of infection.

THROMBOPHLEBITIS.—Thrombophlebitis of the superficial veins usually results from trauma, infection or the injudicious use of sclerosing solutions (Fig. 140). The involved vein becomes firm, tender and cordlike. Swelling and cellulitis of the leg may appear. Varicose veins are understandably susceptible to thrombophlebitis. Conservative treatment is in order. Although the danger of pulmonary embolism is not great, such a possibility must be kept in mind. Extension of the thrombotic process to the deep veins of the leg may lead to the chronically swollen leg of the postphlebitic syndrome.

Fig. 139 (*above*).—Stasis dermatitis and ulceration due to varicose veins. Note pigmentation bilaterally and some chronic edema on the right.

Fig. 140 (*below*).—Acute thrombophlebitis of the saphenous vein in the thigh due to injection of sclerosing solution. Treated conservatively with good results.

related to its development. The lymphatics become dilated, and their valves are rendered functionless. Secondary changes result in the extremity from lymph stasis.

Obstructive lymphedema may result from inflammatory, neoplastic or traumatic agencies. It is relatively frequent after operations for

FIG. 142.—Massive chronic lymphedema of the arm following radical mastectomy for cancer (Elephantiasis chirurgica)

cancer, when removal of large segments of the lymphatic circulation is coupled with the inflammatory reaction of wound healing and/or radiotherapy. This combination results in a decompensation of the lymphatic circulation of varying degree. This situation may occur after radical mastectomy (Fig. 142), radical groin dissection, etc., and may lead to permanent structural changes. The adverse influence of superimposed infection is obvious. It is imperative that edema be minimized

HEMORRHAGE.—Bleeding may occur externally or into the subcutaneous tissues, producing an area of ecchymosis. Bleeding can be easily controlled by elevation of the leg and slight sustained pressure over the bleeding area. Most subcutaneous hematomas will absorb without special therapy. Rarely is specific treatment required for acute hemorrhage from varicosities, but attention should be directed toward their treatment following recovery.

POSTPHLEBITIC SYNDROME.—The postphlebitic syndrome (Fig. 141) is the result of an extensive thrombosis of the deep veins of the leg. It is characterized by chronic enlargement of the leg, usually with pigmentation, induration, ulceration and a variable degree of disability. Varicose veins may or may not be associated. Treatment is directed toward reducing and controlling edema and the complications of tissue anoxia. The operative or the injection treatment of varicose veins associated with the postphlebitic syndrome is contraindicated, except when it can be demonstrated that the deep venous circulation is adequate and that treatment of the superficial veins will improve the circulation of the leg.

LYMPHATIC DISEASE

The flow of lymph is intimately related to fluid flow in other compartments, including the arterial and venous channels and the intercellular spaces. Alterations in one system are reflected by changes in all. For example, chronic venous disease (as well as some arterial conditions) may lead to excessive filtration and inadequate absorption from the capillary loop, resulting in stasis of fluid in the intercellular spaces. The lymphatic channels may be completely overburdened and unable to keep pace with the fluid accumulation in the intercellular spaces. Under these circumstances, secondary effects resulting largely from edema and anoxia of tissues, plus the irritant effect of high protein concentrations in the interstitial fluids, may lead to permanent structural changes. This then becomes a self-perpetuating vicious cycle. The usual changes due to such a waterlogging of the tissues are: edema, thrombotic and fibrotic obliteration of the lymphatics, diffuse fibrosis, eczematoid skin changes, pigmentation, ulceration and permanent limb enlargement.

A congenital type of lymphatic insufficiency which usually involves the legs is known as "Milroy's disease." It is seen most commonly about the time of puberty, and a hereditary factor is presumed to be

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during its early stages; otherwise, permanent and progressive changes can be expected to follow.

The same mechanism is often invoked following extensive soft-tissue or bone injury or primary or secondary malignant disease located at sites where the lymphatic channels are confluent (such as the axilla or the groin), or when severe degrees of venous obstruction lead to the postphlebitic state.

Elephantiasis is the massive enlargement of an extremity due to decompensation of the lymphatic circulation. Filariasis is a classic cause, but the condition may occur after operation (elephantiasis chirurgica). Excision of large segments of subcutaneous tissue, including the dilated fluid-filled lymph spaces and fascia over the muscles (Kondoleon's operation), may provide a basis for the development of new lymphatic communications through deep lymphatics of the muscles, with the restoration of some degree of lymphatic compensation

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ARTERIAL DISEASE

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should be collected and recorded as soon as circumstances permit, because later it may be unobtainable. If the patient is a minor, parental (or legal) permission must be obtained before instituting treatment, except under emergency conditions. A written permit is desirable if surgical treatment under anesthesia is planned.

After emergency measures have been instituted and the examination completed, sequential treatment may be planned. It must be decided whether immediate or delayed treatment of the fracture is in order. Simple fractures, in the absence of complicating injuries, are treated without delay. If associated vascular or nerve injury exists, emergency treatment is mandatory. Open (compound) fractures also require urgent treatment. However, if other serious injuries exist, or the patient's general condition is unsatisfactory, immediate local treatment of the fracture may be contraindicated. Some fractures, such as simple fractures of the skull, are best left alone.

X-ray examination must be performed in every instance. Whether or not x-ray films are secured immediately depends on the condition of the patient. Moving the patient for such an examination may be unnecessary and harmful if his general condition is precarious. Under these circumstances, resuscitive measures have priority and x-ray examination should be deferred until the critical period has passed. If the x-ray examination is delayed, it is advisable that the surgeon record the reasons for doing so, in order to protect himself against the possibility of a subsequent charge of malpractice.

CLASSIFICATION OF FRACTURES

A fracture is a break in the continuity of bone or cartilage, usually due to external violence. Fractures may be classified in many ways, according to location, configuration, time of life, joint involvement, associated bone disease and whether or not they communicate with the exterior. They may result from direct or indirect injury. Fractures may also result from muscle pull, in which case a small piece of bone is avulsed by the force transmitted through the tendon. When rotational forces operate, the fracture is likely to be oblique or spiral in direction. Direct forces usually produce a transverse break. Fractures through diseased bone (pathologic fractures) are most commonly transverse.

In a *closed* (or *simple*) *fracture*, the fracture site does not communicate with the outside. The skin is intact.

CHAPTER 29

Fractures

THE PHYSICIAN who undertakes the treatment of an injured patient assumes a responsibility of considerable magnitude. He must conduct a rapid general examination and, if indicated, institute emergency treatment for those conditions which threaten life. After this, or when the situation is not critical, he may proceed to a more complete evaluation and definitive treatment according to the "four R's": recognition, reduction, retention and rehabilitation.

Despite the fact that the patient may believe he has only a single injury, it is essential that a complete examination be made in order to avoid overlooking associated or other injuries. The record of the initial survey should contain such pertinent items as: the body temperature, pulse and respiratory rate, blood pressure and responsiveness, as well as a description of the general and local physical findings. These data should be concisely, but accurately, noted.

The local examination is likely to demand the greatest attention Not only must the broken bone be assessed; but the condition of the vessels, nerves, tendons and muscles of the injured part must also be carefully noted. The associated injuries to major nerves or blood vessels are often more important than the fracture itself. It is essential that the examiner be familiar with tests for the integrity of the major nerves and vessels Observations relating to the local circulation and innervation should always be recorded.

In most fracture cases the history is not complicated. For completeness of the record, such items as the time, place and circumstances surrounding the accident should always be included. Whatever previous treatment the patient has received should be detailed. Usually the patient will be able to give the history, but sometimes it must be obtained from relatives or other interested parties. This information

deposition of calcium salts, with formation of a callus and ultimate bone production. This process is considered in Chapter 2, on Wound Healing and the Care of Wounds.

The rate at which uncomplicated, or normal, fracture healing occurs cannot be accelerated. However, local or systemic disturbances may interfere with the healing process. The most important adverse influences are:

1. Lack of reduction (or of "setting the broken bone"). The bone ends are not in contact. Distraction, or separation of the bone ends, as well as over-riding, may delay or prevent healing.
2. Failure of immobilization. Like other tissues, bones heal best while at rest. Early in the course of fracture healing, immobilization is needed in all planes. Later, longitudinal stresses may stimulate union.
3. Loss of the fracture hematoma. This results in loss of the scaffolding necessary for the formation of granulation tissue and callus which binds the bones together.
4. Foreign bodies in or near the fracture site. Foreign bodies may slow the rate of healing. Under certain conditions, metallic appliances are used for internal fixation. The advantages which they provide in terms of reduction and retention may outweigh their disadvantages.
5. Infection at the fracture site. Infection prolongs the healing time of bone, the same as in other tissues.
6. Poor blood supply to one or both fracture fragments. This impairs the healing process or makes it impossible.

A fracture is said to be "healed" when immobilization is no longer needed. Some fractures heal in a few weeks; others require months, or years. Complete healing, including final realignment and maturation of bony trabeculae, is a process which continues for a long time. The state of the healing process may be judged by the time interval since the fracture occurred, the local findings and the x-ray appearance.

The time required for fracture healing varies according to the location, type, blood supply, complications, treatment used and, to some extent, the age of the patient. There is no marked difference in the rate of fracture healing between youth and old age, but it is much more rapid in infancy and childhood than in adult life.

When examination reveals no motion at the fracture site, no local pain on movement and the callus is nontender, firm and contracted, the fracture has probably healed.

Healing has probably taken place when, in the x-ray film, the

In an *open* (or *compound*) *fracture*, the fracture site communicates with the outside. An open fracture may result from an external injury which produces a wound that enters the fracture site; from an internal injury (from bone ends) which penetrates through the tissue, including the skin, to the outside; or from a combined external and internal injury.

Birth fractures occur in the newborn as a result of birth injury. The humerus is most commonly fractured; the femur and clavicle less often Such fractures heal rapidly.

A *greenstick fracture* is a type of fracture that occurs in children and is likened to the greenstick because only one cortex is broken while the other remains intact. Frequently there is a longitudinal crack extending both ways from the incomplete transverse fracture line There is usually little deformity, and healing is rapid.

An *epiphyseal separation* may be considered a fracture. It occurs in children when the epiphyses are open. Actually, it consists of a fracture through the epiphysis.

In an *impacted fracture*, the ends of the fractured bone are driven together and become locked.

In a *comminuted fracture*, there are more than two bone fragments.

In a *complicated fracture*, there is associated damage to important vessels, nerves or other structures.

A *fracture dislocation* is a fracture and a dislocation in the same region—for example, dislocation of the shoulder associated with a fracture of the humeral neck.

A *pathologic fracture* is a fracture through a bone weakened by infection, tumor or osteoporosis. Usually the force which produces such a fracture is negligible, compared to that required to break a normal bone.

A fracture is *complete* or *incomplete* according to whether the break extends through all or part of the cortex.

In a *displaced fracture*, the fragments are in abnormal position. In an *undisplaced fracture*, they are in normal position.

Transverse, *oblique* and *spiral* are terms used to describe the configuration of the fracture line.

HEALING OF FRACTURES

The healing of a fracture is substantially the same process as that which occurs in the healing of other tissues, except that there is a

Fluoroscopic examination generally should be avoided. It usually affords little information, provides no permanent record and is potentially dangerous to both the patient and the examiner.

Adequate x-ray films in *two* planes (anteroposterior and lateral) are required (Fig. 143). Stereoscopic films are sometimes necessary. Because the possibility of injury beyond the obvious fracture site often

FIG. 143.—X-ray films showing fractures of the lower end of both bones of the forearm with displacement (Colles' fracture).

exists, the joints above and below the fracture are usually x-rayed. It may be advisable to take films of the opposite, the normal, extremity for comparison with the injured side.

With experience, interpretation of the films is usually not difficult. The fracture line may be obvious. The degree and type of displacement can be determined by studying both views. In some cases the fracture line is obscure but becomes evident when the films are studied for disturbed relationships, irregularity of the cortex or disruption of the normal cortical pattern. Occasionally the fracture line cannot be visualized in the initial x-ray films, but becomes evident after bone absorption has occurred. For example, if a patient has a suspected fracture of the carposcaphoid but the x-rays are negative,

fracture line is no longer clearly visible (obliterated) and the callus has become dense from new bone formation. If there is sclerosis of the bone ends and little or no callus, impairment of the blood supply, and possibly nonunion, must be suspected.

In general, then, the fracture may be said to be healed when sufficient time has elapsed for that particular type of fracture to have healed; when there is evidence of stability at the fracture site, when the callus is contracted and not tender; and when the x-ray findings reveal secure bony union.

DIAGNOSIS OF FRACTURES

The diagnosis of fracture is based on the history, physical examination and x-ray studies.

The patient should be questioned regarding the mode and severity of the injury. This may give an indication of the type of fracture sustained. Often the patient may state that he heard or felt a snap as the bone was broken. For example, it is common for an elderly patient who has tripped on a rug to feel a snap in the hip and then fall to the floor with a hip fracture. There may be pain, swelling, "grating" and disability immediately following the injury. Examination will reveal:

1. Localized tenderness over the fracture site.
2. Ecchymosis and swelling due to hemorrhage and edema.
3. Deformity. Certain fractures produce a characteristic change; e.g., "silver fork" deformity of Colles' fracture.
4. Crepitus The grating produced by the two ends of the bone rubbing against each other on movement is characteristic. The sign may be noted by the patient. Do not test for crepitus, because there is danger of causing more damage.
5. False motion. False motion definitely indicates the presence of a fracture. It should be searched for with extreme gentleness.
6. Loss of function.

X-ray examination is mandatory for all fractures and possible fractures. Generally, fracture cannot be ruled out by any other means For this reason, if, after injury, a fracture is possible, the surgeon must proceed on the assumption that there is a fracture until it is ruled out. This aspect of medical practice has many legal implications, and from the viewpoint of protecting both the patient and the physician, x-ray examination must be done.

after ten to fourteen days of appropriate treatment the clinical diagnosis may be verified by the findings on x-ray examination.

The examiner must avoid confusing accessory bones, such as the sesamoids, with fractures. The sesamoids are common in the hands and feet, but they also occur in the elbow, knee and other areas.

X-ray films should be made (1) before treatment, (2) after reduction and/or after immobilization and (3) throughout the healing period at frequent intervals.

The pertinent items to be checked before definitive treatment is

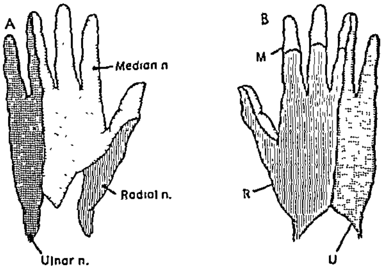

FIG. 145.—Sensory innervation of the hand. A, the palm of the right hand. B, the dorsum of the right hand. (Note: There is considerable variation and overlapping of sensory areas.)

undertaken, as recommended by the American College of Surgeons in the *Outline of the Treatment of Fractures* (1954), are:

1. The direction of the plane of fracture
2. The amount of comminution
3. The amount and character of the displacement
4. The amount and character of injuries to the adjacent tendons, muscle and ligaments
5. Whether there has been an associated nerve injury [see, e.g., Figs. 144 and 145]
6. Whether a second fracture exists, either adjacent or remote
7. Whether a dislocation at the adjacent joint complicates the fracture
8. Whether the circulation at or distal to the site of injury has been seriously compromised

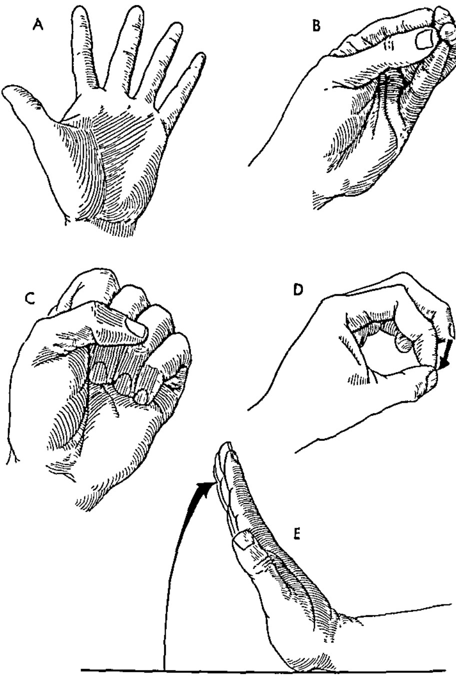

FIG. 144.—Some simple tests of nerve and tendon function of the hand. *A*, complete extension of the fingers and thumb, for testing function of the median, ulnar and radial nerves and the extensor tendons. *B*, "Make a cone," for testing function of median and ulnar nerves and the intrinsic muscles of the hand. *C*, "Make a fist," for testing function of the median and ulnar nerves, flexor tendons of fingers and adductor muscles of the thumb. *D*, "Make a circle" with the thumb and index finger, for testing function of the median and ulnar nerves and muscles of these fingers. *E*, "Raise the hand" from table, for testing the integrity of the radial nerve and extensor muscles of the fingers and wrist

a splint. Morphine and related drugs must be used sparingly and judiciously. That they often compound the difficulties of management of severely injured patients, particularly those with head, chest or abdominal injuries, has already been emphasized.

The injured patient should be reassured and made as comfortable as possible. He should be kept warm. He must not be subjected to further mental or physical stress. Additional injury must be prevented at all costs. Closed fractures can be converted into open fractures by rough handling. Extensive damage to major vessels and nerves sometimes results from unnecessary manipulation or failure to splint the fractured extremity.

"Splint 'em where they lie" is a rule to remember. For the extremities, pillows, boards, etc., tied into place, may be used. The Thomas splint, if available, is best for fractures of the lower extremity. Traction may be used if indicated. Satisfactory immobilization of the leg may often be achieved by placing a pillow or folded blanket between the legs and binding them together. The well leg then serves as a splint for the injured leg. For upper extremity injuries, the arm may be immobilized in a sling or swath bandaged to the chest wall.

Patients with vertebral injuries (Fig. 146) should be handled and transported in "one piece." Flexion must be avoided, and the patient should be moved only after adequate preparations have been made. He should be gently rolled, or lifted, in "log fashion" into the prone position onto a firm surface, such as a door. When moving the patient, there must be sufficient help available to keep the body straight at all times. For cervical spine injuries, the patient should be placed face up, with sandbags or blankets placed at each side to prevent rotation and flexion of the head on the neck.

Objectives in Treatment of Fractures

Often the most urgent need is to save the patient's life, which may be threatened by hemorrhage and shock, respiratory or cardiac failure, or infection, including tetanus or gas gangrene. First, the patient himself must be treated. Next, the surgeon may consider measures to provide early healing and return of function. Finally, cosmetic results may be considered. At all times, the surgeon must be sure that treatment will not make the patient worse. A few examples will illustrate these principles:

1. A middle-aged man saw his physician for an ankle injury of two

TREATMENT OF FRACTURES

Emergency (First-Aid) Treatment

The first-aid treatment of fractures includes those measures which are applied at the scene of the accident and during transport. It does

Fig 146 — Compression fractures of the vertebral column Note the wedging of the 12th thoracic and 1st lumbar vertebrae Damage to the spinal cord resulted in paraplegia

not differ greatly from that given in the emergency room of a hospital, but it is necessarily more limited.

The immediate objectives are: to sustain and support life and to avoid further injury Respiratory obstruction, hemorrhage, shock and infection are high-priority considerations. Pain must be relieved. This may be accomplished by simply immobilizing an injured extremity in

A figure-of-eight elastic bandage would have given a better result. Bed rest with support between the scapulae, plus lateral traction, gives excellent cosmetic results. Open reduction of the clavicle is rarely indicated except to relieve pressure on neurovascular structures or to prevent sharp bone spicules from perforating the skin and producing an open fracture.

5. An elderly woman with an intertrochanteric femoral fracture was treated with a double-hip plaster cast (spica). The fracture healed in good position, but pressure from the cast and inadequate nursing care led to pressure sores (decubitus ulcers) over both anterosuperior iliac spines and the sacrum.

Intertrochanteric fractures of the femur almost always heal without difficulty, regardless of whether the fracture is reduced or not. Treatment either by traction or by internal fixation would have been preferable in this instance.

6. A soldier was injured in an automobile accident and experienced severe back pain. He moved his feet well but did not attempt to stand up. Sympathetic passers-by lifted him by his shoulders and feet, severely flexing the trunk. From that moment, the patient was paraplegic.

The physician is seldom implicated in such a tragedy. He learns to roll or lift the patient log-fashion; to apply shoulder (or head) traction when moving him; and to transport him with the face down in case of dorsolumbar spine injury, or supine with the head extended in cervical spine injury.

The surgeon must always consider how the proposed treatment would benefit the patient. Mild compression fractures of the lumbar spine and all compression fractures of the dorsal spine, without neurologic deficit, are best left impacted without attempted reduction. These impacted fractures heal more quickly when left impacted. They permit the patient to become ambulatory quickly; muscle strength is retained; and functional recovery hastened. If ambulatory immobilization of the spine is needed, a few weeks after the injury a well-fitting plaster jacket or brace may be applied.

Nasal fractures may be treated (*a*) to improve the nasal airway and (*b*) to improve the appearance. Malar fractures may require elevation into proper position (*a*) to prevent an unsightly sunken cheekbone or (*b*) to correct vertical diplopia due to orbital floor depression, with pull on the lateral canthal ligament. Mandibular fractures are treated primarily to restore normal dental occlusion; hence

weeks' duration. The x-rays showed a fracture of the tip of the medial malleolus with moderate separation of the fragments but no significant displacement. Open reduction and fixation with a metal screw was carried out. Postoperative infection occurred and was followed by a prolonged illness. A year later there was persistent wound drainage, the joints were stiffened, and the tibia was sequestrating (osteomyelitis).

While operation may actually have been indicated in this particular case, the ensuing catastrophic complications point up the risks involved

2 A young man suffered an open (compound) fracture of the tibia The wound was débrided; a heavy metallic plate was used to fix the fragments; and the skin was left open, with the plate exposed in the infected wound. When seen six months later, the visible bone was dead-white and devoid of periosteum. Pain was severe. The patient demanded that the extremity be amputated. The request was refused because of the patient's youth and the intact blood and nerve supply and because measures such as removal of the foreign bodies, prolonged immobilization, antibiotic therapy and general supportive measures had not been attempted. The patient left the hospital to search for someone who would amputate the leg.

Internal fixation is used in certain open fractures only when there is little chance of infection, and then only if there is a particular need for internal fixation. When internal fixation is used, the skin must be closed over the appliance.

3. An elderly patient with an impacted fracture of the neck of the humerus was treated for several weeks in a shoulder cast. The fracture healed well in its original, satisfactorily impacted position. The shoulder remained stiff and painful following removal of the cast, and motion at the elbow was also restricted as a result of prolonged immobilization

Humeral neck fractures in patients beyond middle age are usually treated with a sling or a light "hanging cast." Shoulder exercises are encouraged from the beginning, to prevent shoulder stiffening Impacted fractures of the humeral neck ordinarily require only a sling.

4. A young girl with a fractured clavicle was treated by open reduction in the hope of obtaining the best possible cosmetic result. The clavicle bowed at the site of insertion of the screw; excessive callus formed a bulge beneath the skin; and an unsightly operative scar ran along the clavicle. The scar was tender, probably from postoperative neuromas of divided supraclavicular nerves.

of reduction of the fracture were the only consideration and if the *bone*, rather than the whole patient, required treatment, a cult of "bone carpenters" would be fully justified.

Methods of Reduction and Immobilization

Manipulative Reduction.—During manipulative reduction the x-ray films should be in view. Anesthesia should be consistent with the relaxation necessary for manipulation and for prevention of pain.

A displaced fracture may commonly be restored to normal alignment by the application of *longitudinal traction combined with the* indicated angulating or rotating forces. In some fractures, it is first necessary *to increase the deformity and to disengage the fragments*, or to bring them into marginal contact. The next step consists in correction of alignment. The forces required to effect manual reduction are, in general, the reverse of the forces which produced the fracture.

Consideration must always be given to the direction of muscle pull on the fragments when reducing a fracture. Usually, the position of the proximal fragment is accepted and not altered, while manipulation is directed toward bringing the distal fragment into alignment with the proximal fragment. This is necessary because the surgeon can ordinarily control the position of only the distal fragment.

Traction Reduction.—Traction is used to overcome over-riding or for regaining normal length. It may be applied to an extremity by means of adhesive strips (skin traction) or by metal wires or pins placed through bone (skeletal traction). A system of ropes, pulleys and weights is constructed according to the forces needed. Continuous traction may be used for days or weeks, for purposes of both reduction and immobilization. The amount of traction can be altered, according to need, by changing the amount of weight used.

Bryant's traction is a type of overhead traction usually transmitted through adhesive strips to the skin of both lower extremities. It is used to reduce and immobilize fractures of the femoral shaft in infants and young children. Only sufficient weight is applied to lift the buttocks slightly off the bed. With this method the child is comfortable and nursing care is facilitated.

Skeletal traction is preferred in adults because their skin will not tolerate the pull necessary to effect reduction. The usual site for the introduction of a metallic pin or wire (Steinmann pin or Kirschner wire) in the lower extremity is through the tibial cortex deep to the

the teeth must be held in proper occlusion during the healing of the fracture.

Ankle fractures are treated to restore normal relationships in the ankle mortise and to avoid irregular joint surfaces. Os calcis fractures are treated so that (*a*) ankle motion is uninhibited; (*b*) joint surfaces are as smooth as possible, to lessen the severity of post-traumatic arthritis; and (*c*) the heel is sufficiently restored in contour and width to permit the wearing of shoes. Patellar fractures are treated (*a*) to restore functional continuity of the quadriceps mechanism; (*b*) to prevent the development of a rough patellar surface which articulates with the femur, and (*c*) to remove material, such as bone fragments, from the knee joint.

Rib fractures may be treated by taping and by using intercostal nerve blocks to relieve painful respiration and to enable the patient to clear the respiratory passages by coughing. With multiple rib fractures at two or more sites, the chest wall may become unstable, producing paradoxical chest movement and marked interference with ventilation. In this circumstance, stabilization of the mobile chest wall by means of external fixation may be necessary.

Femoral shaft fractures are treated primarily to prevent angulation, foreshortening or rotary malalignment They usually heal well, but it is important to prevent deformities which might cause limp and back strain from shortening, knee strain from angulation, or an awkward walking position ("toe in" or "toe out") from rotation.

Certain fractures associated with a poor blood supply to one fragment (e.g., femoral neck and carposcaphoid) require nearly anatomic reduction with complete and prolonged immobilization, so that the blood supply from the well-vascularized fragment can quickly "invade" the devascularized fragment.

Stable fractures are those in which closed reduction is easily maintained as a result of favorable muscle pull or of fracture ends that were well formed for locking. These fractures are best treated by closed reduction and the application of an appropriate cast. *Unstable* fractures are usually oblique and subject to a shearing force from muscle pull or functional stress. In unstable fractures the surgeon must provide against displacement by the use of traction to counteract muscle pull or to secure the fragments against redisplacement by internal fixation.

The foregoing discussion regarding fractures illustrates the necessity for careful consideration of the objectives of treatment. If beauty

of reduction of the fracture were the only consideration and if the bone, rather than the whole patient, required treatment, a cult of "bone carpenters" would be fully justified.

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inferior portion of the tibial tubercle. While traction applied here has the disadvantage of pulling across the knee joint and stretching its capsule and ligaments (as in femoral fractures), the alternative position, above the femoral condyles, has equally important disadvantages.

Skeletal traction may also be applied through the lower tibia, os calcis, greater trochanter of the femur, olecranon, distal radius and the bones of the hand and foot.

Crutchfield tongs are used when traction is indicated for cervical fractures or dislocations. They are similar to ice tongs but have a set-screw adjustment to lock them into position after the prongs have been inserted into the outer table of the skull. Traction is applied through them to the skull and the cervical spine.

Internal Fixation.—Fractures may be reduced and fixed by open operation. A variety of metallic contrivances, including plates, screws, nails, bolts, bands, wires and intramedullary pins are used. Stainless steel is most popular at the present time. The metals implanted should be those that are least irritating to tissues, and all of the metallic parts should have the same chemical composition in order to avoid the "battery effect" of dissimilar metals, which may lead to impaired healing.

External-Internal Fixation.—The term "external-internal fixation" was coined to cover a variety of technics which involve external fixation of metallic pins placed into, or across, the fractured bone. The pins may be inserted through the bone to project from the skin on either side of the extremity. One or two such pins placed above and below the fracture site can be stabilized externally by use of adjustable metal bars or by incorporating the pins into a rigid plaster cast. This method of fixation is not widely used today.

Insofar as possible, joints should be immobilized in a position of function. For the hand, this means slight dorsal flexion at the wrist and flexion at all finger joints. In this position, capsular and ligamentous shortening is minimized and restoration of function aided, as compared to the course following immobilization of the hand in extension. By preference, the elbow is immobilized at 90 degrees flexion; the forearm in mid-pronation-supination; and the shoulder in moderate abduction and forward flexion The ankle should be immobilized at 90 degrees with neither inversion nor eversion. The knee may be placed in complete extension or slight flexion. The hip is most advantageously held in slight flexion and moderate abduction.

Open Reduction.—In certain fractures, reduction and retention can be achieved only by open operation. Operation should be undertaken only if the anticipated result is definitely better than can be expected from closed treatment. The decision to operate must often depend on the experience and judgment of the surgeon. The following conditions favor open reduction:

1. Open fracture; fracture site already exposed (usually no internal fixation is added)
2. A fracture in which closed reduction is known to usually fail
3. An unstable fracture which is likely to become displaced
4. A fracture in which there is evidence of interposition of soft tissues between the fragments
5. A fracture in which there are loose fragments in a joint
6. A fracture in which improved immobilization of the fragments is desirable (e.g., femoral neck fractures)
7. Certain fractures in which early mobilization of the patient is desirable
8. Certain pathologic fractures
9. To lessen the period of hospitalization or absence from work
10. To facilitate cast care (e.g, intramedullary nailing of the femur)

The *objections* to open reduction are:

1. It usually entails a major operation.
2. There may be serious interference with the blood supply to the fracture site.
3. There is danger of infection.
4. There may be loss of the fracture hematoma.
5. The bone ends may be held apart by the apparatus used.
6. Even after internal fixation, a plaster cast is usually necessary.
7. Periosteal stripping and dissemination of the hematoma may result in development of myositis ossificans.
8. Important vessels and nerves near the fracture site may be injured.
9. The introduction of foreign bodies favors the development of infection.
10. Metallic objects in or near a joint increase the likelihood of joint stiffness.
11. In children, damage to the epiphyses alters growth of bone.

Treatment of Open (Compound) Fractures

The term "open fracture" is preferred to the older designation, "compound fracture." In open fracture the fracture site communicates with the exterior. The compounding may result from within, as when a bone spicule punctures the overlying skin, or from without, as when an external force produces a fracture and a wound through all tissues overlying the bone. Bacterial contamination depends on the extent of the wound, the circumstances of the wounding and whether foreign material has been carried into the wound. Open fractures are generally considered "contaminated" from the start. During the first eight to twelve hours following injury, careful wound treatment, including débridement, may serve to convert the wound into a relatively clean or "uncontaminated" state, so that it may be closed. The fracture may then be treated as a simple fracture, except that antibiotic protection is added. Open fractures should be considered infected if twelve (or more) hours have elapsed before treatment is instituted. The time factor is variable according to the circumstances. The surgeon must be certain to use good judgment in deciding on the treatment of open fractures.

The following examples will illustrate how circumstances may cause deviation from general rules:

1. An open fracture, compounded from within and sustained in the home, could be considered "contaminated" rather than "infected," although not treated until eighteen hours after the injury. During the interval the small wound was covered with a sterile dressing, and the skin and clothing at the site of compounding were clean.

2. A farmer suffered an extensive leg wound when he was run over by a tractor in the barnyard. The tibia was fractured, and manure-covered clothing had been driven deeply into the wound. A dirty handkerchief was packed into the wound to control bleeding Treatment in the hospital was instituted within four hours after injury. After careful débridement and irrigation, the wound was not closed. The surgeon treated the wound as if it were infected, although the theoretical "golden period" had not passed. Furthermore, the surgeon administered generous prophylactic doses of tetanus and gas gangrene antitoxin, as well as antibiotics.

3. A college student struck a man in the mouth, thereby suffering a knuckle laceration and an open metacarpal fracture. Treatment was instituted within two hours. The surgeon cleansed and débrided the

wound but left the skin open. The fracture was reduced and immobilized. The patient was hospitalized, and antibiotics and tetanus antitoxin (3,000 units) were given. The surgeon considered that there was danger of serious infection from "human bite wounds," and he believed that this danger outweighed the advantage of following the principle of "débride and close hand wounds in order to obtain the greatest degree of functional recovery."

Only in exceptional circumstances should internal fixation appliances be used in open fractures. The introduction of foreign materials into an area of contamination favors the development of infection and prolongs the course of established infection. The placement of *appliances tends also to disseminate infection and to cause injury to* adjacent normal structures, making them more vulnerable to infection.

Some generalizations applicable to the treatment of open fractures may be listed under "initial" and "definitive" categories, as follows:

A. *Initial treatment (in emergency room):*

1. Stop hemorrhage and treat shock.
2. Splint the fracture and cover the wound.
3. Quickly check for associated injuries.
4. Quickly check for fracture complications, including vascular and nerve damage.
5. Relieve pain. If needed, give morphine or Demerol® in small doses intravenously.
6. Obtain indicated x-ray films if the patient's condition permits.
7. Administer tetanus antitoxin or tetanus toxoid, or both. Gas gangrene antitoxin is indicated in certain cases.

B. *Definitive treatment (in operating room):*

1. Cleanse, shave and drape the wound region.
2. Irrigate the wound with copious amounts of saline, from within outward Remove foreign bodies; excise devascularized and damaged tissues. Open fascia widely to permit exposure of deeper recesses of wound. Remove small detached bone fragments; rongeur away bone which has dirt ground into it.
3. Irrigate the wound repeatedly with large amounts of saline until fat globules, blood clots, dirt fragments and all loose tissue has been completely removed. Obtain hemostasis with pressure and fine catgut ligatures.
4. Reduce the fracture under vision.

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B. *Late treatment (chronic localized infection):*

1. Immobilize the fracture.
2. Give tetanus antitoxin or tetanus toxoid.
3. Remove sequestra.
4. Administer appropriate antibiotics.
5. Correct anemia and nutritional deficiencies.

The foregoing measures are those that are used in treating acute or chronic osteomyelitis of a localized type. If nonunion results, a bone-grafting procedure may be necessary when the infection has cleared and all wound drainage has ceased for a period of several months. It should be remembered that tetanus toxoid or tetanus antitoxin is indicated, not only at the time of the original injury (open fracture), but also before late manipulation or operation on the fracture. There is always the possibility of activating the growth of tetanus spores in the wound.

GENERAL COMPLICATIONS OF FRACTURES

The three main causes of death from fractures are: shock, which may kill within the first few hours after injury; fat embolism, which may kill during the first week after injury; and thromboembolism (pulmonary embolism), which does not often cause death until the second or third week after injury.

Shock and Hemorrhage.—Shock and hemorrhage may endanger the patient's life in the early period following injury. Traumatic shock is best treated by splinting, by relieving pain, by avoiding additional injury and by the judicious replacement of depleted blood volume. If the response is unsatisfactory, search for hidden injuries.

Fat Embolism.—Fat embolism occurs following multiple fractures and fractures of large bones, particularly in elderly patients with fatty marrow cavities. It is more common than realized and is often overlooked, especially in its milder forms. The symptoms and signs which may appear a day or two following injury are due to fat globules in the capillaries of the lungs, brain, heart, retina, skin and other organs. There may be fever and tachycardia. There is often dyspnea, with cyanosis and cough. Retinal and punctate skin hemorrhages may be found In severe cases, reflexes are lost and convulsions occur. Mental confusion appears, and the patient may become comatose and die.

Fat globules can often be demonstrated in the urine and sputum. No satisfactory treatment has been developed. Rough or repeated manipulation of fractures predisposes the patient to fat embolism.

5. Immobilize the fracture by—
   a) Cast,
   b) Cast incorporating Steinmann pins, proximal and distal to the fracture site,
   c) Skeletal traction or
   d) Internal fixation—*only* in exceptional circumstances and *only* when the wound can be safely closed.
6. Close the wound or leave it open, depending on—
   a) Nature of compounding,
   b) Degree of contamination,
   c) Time interval since injury,
   d) Completeness of débridement,
   e) Ease of closure (do not close under tension) and
   f) Circulatory status (do not close if circulation is impaired).

C *Contraindications* to the closure of an open fracture wound are:
1. Wounds that have been open eight to twelve hours
2. Badly contused wounds
3. Gross wound contamination
4. Barnyard and bite wounds, etc.
5. Necessarily incomplete débridement (vessels and nerves in the wound prohibit complete débridement)
6. Closure impossible without tension
7. Impaired circulation

Treatment of Infected Fractures

The treatment of fractures that have established infection may be considered under the headings of *early* and *late*. The appropriate measures to be taken in each case are given below:

A. *Early treatment (acute localized infection):*
1. Institute general measures against infection, such as—
   a) Rest,
   b) Tetanus antitoxin (or toxoid) and gas gangrene antitoxin, if indicated,
   c) Antibiotic therapy (consider locally used drugs) and
   d) Maintenance of metabolic needs.
2. Institute local treatment of infection:
   a) Provide wound drainage.
   b) Immobilize the fracture.
   c) Remove foreign bodies or sequestra.

where the blood supply to one of the fragments is poor, accurate reduction and prolonged immobilization are necessary for healing. Generally, union is slower in the midshafts of long bones, where the cortex is dense and the bone is relatively poorly vascularized, than in the ends of long bones and flat bones, where the bone is cancellous and highly vascularized.

In *nonunion*, the fracture has not united in spite of an elapse of time believed sufficient for healing. There is false motion (unless the fragments are held by a fixation apparatus), and the x-rays show a rounding-off of the sclerotic bone ends with obliteration of the marrow cavity. The situation is such that healing will not occur despite continued treatment. Established nonunion requires open operation and usually bone grafting for its correction. Prolonged immobilization and compression forces will not bring about healing but will tend to encourage the development of a pseudoarthrosis, or false joint, at the fracture site.

In certain locations, bone fragments may be fixed by fibrous tissue instead of by callus and mature bone, even though the fragments are viable and have an adequate blood supply. An example of such healing is seen in the *fibrous union* which occurs following fracture of the tip of the medial malleolus of the tibia. The functional result may be satisfactory; and because the motion between fragments is slight, a pseudoarthrosis does not develop.

Causes of Delayed Union and Nonunion

Several causes of delayed union and of nonunion are described below.

Poor Blood Supply to One Fragment.—Poor blood supply is likely to interfere with healing in femoral neck fracture, where the proximal fragment has a poor or absent blood supply; in carposcaphoid fracture, where the proximal fragment may be deprived of an adequate blood supply, in the lower third of the tibia, where the distal fragment is relatively poorly vascularized; and in fracture of the astragalus, where the posterior fragment is likely to be separated from its chief blood supply.

Poor Reduction.—Accurate reduction of the fracture is required for healing of the tibial shaft, the humeral shaft, the femoral neck, and other similar bones. The poorer the blood supply, the more important does accurate reduction become. Bones which have a rich

PNEUMONIA.—Pneumonia is most likely to appear in the elderly patient who is immobilized and inactive in bed. Absolute bed rest should be avoided whenever possible. Good nursing care with frequent changes in the patient's position, as well as the measures advocated for keeping the tracheobronchial tree free of secretions, do much to lessen the danger of "hypostatic pneumonia." Antibiotics are of value, but they do not insure against pulmonary complications or obviate the need for good medical and nursing care.

DECUBITUS ULCER.—Decubitus ulcer is likely to appear in elderly patients. Immobility, lack of cleanliness, moisture, sustained pressure and inadequate nursing care usually can be incriminated. When a decubitus ulcer has developed, the problem, to nurses and doctors, becomes one of "more care required to affect healing than would have been required for prevention." The treatment of an established decubitus ulcer is a complicated task.

URINARY CALCULI.—Urinary calculi sometimes appear in the immobilized patient owing to an increased excretion of calcium from disuse, decalcification of bones. Urinary stasis, a low urinary volume and alkaline reaction favor calcium precipitation The prevention of stones requires: avoidance of prolonged immobilization, a high liquid intake and output and perhaps an "acid ash" diet.

DELAYED UNION AND NONUNION

In *delayed union,* the fracture has not healed at the time when similar fractures would ordinarily have healed. The term implies that healing is likely to occur with continued treatment. The x-ray films may show scanty callus or a persistence of the fracture line. There may be a distinct gap between bone ends owing to absorption, especially if rigid internal fixation material (appliances such as plates) has served to hold the bone ends apart. There may be some sclerosis of the bone ends, but not severe sclerosis and rounding-off of bone ends. Clinically, slight motion is likely to be demonstrable at the fracture site. Delayed union may be treated by prolonging the period of immobilization. Often the institution of a compression force (while avoiding rotational and lateral stresses) hastens healing. Delayed union occurs frequently in simple fractures in the lower third of the tibia if either reduction or immobilization are not good. In this area a reduced blood supply to the distal tibial fragment slows the healing time. If complicated by infection, union may be delayed for many months. In any fracture

Enzymatic disruption of granulation tissue and callus slows the reparative processes. Small bone fragments which ordinarily become incorporated in callus die and cause prolonged wound drainage. Hyperemic decalcification persists for a long time, and calcification of the callus is delayed until the infection is controlled.

ASEPTIC NECROSIS OF BONE

Aseptic necrosis of bone is the result of the loss of blood supply which may follow fracture or dislocation. The dead bone may be gradually absorbed and replaced by new bone from the adjacent bone (creeping substitution), or it may be absorbed or sequestrated. The adjacent joint surfaces may undergo osteoarthritic changes.

Aseptic necrosis frequently occurs after femoral neck fractures, where there is loss of blood supply to the head of the femur and failure of revascularization. It also may follow fracture of the carposcaphoid or posterior dislocation of the hip.

After injury, devascularized bone generally retains its normal density, while the adjacent vascularized bones undergo traumatic hyperemic decalcification (osteoporosis). Hyperemic decalcification may also occur as a result of infection. This leads to a "paradoxical increased density" of the devitalized bone. The density (due to calcium content) of the dead bone is not increased at all but is merely not changed because the blood supply necessary for decalcification is absent. Hence, in x-ray films the sequestra in osteomyelitis appear dense, as compared with surrounding bone Similarly, if the femoral head undergoes necrosis after femoral neck fracture, it will appear dense when compared with the adjacent bones, which have become less dense. When a severe degree of osteoporosis exists before the fracture, the expected "paradoxical increased density" may not become evident in the devitalized bone. Because "death" of bone is judged by the bone's relative density, it is necessary to wait two to three months after femoral neck fracture before an opinion regarding the status of the blood supply of the femoral head is valid.

PATHOLOGIC FRACTURES

Pathologic fractures (Fig. 147) are those produced in bones weakened by generalized or local disease. They usually result from injuries considered insufficient to fracture normal bones. A pathologic condi-

blood supply to both fragments require little more than some degree of contact between the fracture surfaces for solid healing.

Improper Immobilization.—Inadequate immobilization results in continued motion at the fracture site, and callus is broken up as fast as it is laid down. Osseous union is thereby delayed or prevented. Inadequate casts may account for inadequate immobilization. A short leg (below-the-knee) cast, applied for a fracture of the tibia, might lead to this result. The principle of immobilization which states that the joint above and the joint below the fracture site must be restricted would be violated by the use of a short leg cast. Too brief a period of immobilization, as from too early removal of a cast, will have the same effect The failure of internal fixation material will also allow motion to occur and delay healing.

Interposition of Soft Tissue.—Tissue between the fracture ends, such as capsule, tendon, muscle or fascia, delays healing and may prevent healing altogether. Sometimes muscle strands caught in a fracture site, which is otherwise reduced, will undergo necrosis and, like the fracture hematoma, will be converted to granulation tissue, callus and finally bone. In other instances, such interposition may allow continued motion and prevent bony healing.

Distraction of Fragments.—This condition might be considered a "failure of reduction," but it justifies separate consideration. Distraction is not a problem when fracture treatment consists of closed reduction by manipulation and immobilization by cast, because the muscles hold the bone ends together throughout the healing period. When however, internal fixation is used, the bone ends may be held rigidly (but slightly) apart from the beginning; or if absorption of the bone ends occurs, the ends are held slightly separated by the appliance. Healing is then greatly delayed or impossible. Similarly, whenever a fracture is immobilized in traction, care must be taken to avoid distraction for any period of time.

Loss of Fracture Hematoma.—All other factors being equal, fractures heal best when the fracture hematoma is left undisturbed. The hematoma provides a medium for calcium deposition and a local source of calcium, and it also probably contains osteoblasts from fragmented periosteum and endosteum. In some circumstances, however, the advantages gained by open reduction may outweigh the disadvantages of disturbing the fracture hematoma.

Infection at Fracture Site —An infection at the site of fracture slows healing, but infection in itself does not usually cause nonunion.

B. *Local diseases:*
1. Congenital bone cyst
2. Primary malignant disease of bone
3. Metastatic malignant disease of bone
4. Osteomyelitis
5. Localized bone atrophy (e.g., poliomyelitis)
6. Rickets

The principles of treatment are similar to those applicable to other fractures, but there is a tendency to use internal fixation more frequently. When possible, the underlying disease which leads to the fracture should also be actively treated.

COMPLICATED FRACTURES

Complicated fractures are those which are associated with injury to blood vessels, nerves, and other soft tissues. Examples of local complications include the following:

Local Hemorrhage.—External bleeding is controlled by measures previously described. Concealed bleeding, particularly after fracture of the femur, may be sufficient to cause the patient to go into shock. Rib fractures may lacerate intercostal arteries and produce exsanguinating hemorrhage into the pleural cavity. The control of external hemorrhage by wound packing has the advantage of avoiding further injury to the major artery. The tourniquet should be avoided, if possible.

Traumatic Arterial Spasm and Thrombosis.—Localized trauma may result in segmental arterial spasm. It is sometimes severe enough to justify surgical exposure for relief of local pressure and vasospasm. Stripping of the adventitia and sympathetic blocks are also used.

Severe contusion of an artery may result in thrombosis. Thrombosis may cause widespread reflex vasospasm with acute arterial insufficiency. Sympathetic blockade may be of considerable aid in releasing spasm of the collateral vessels. Thrombectomy, excision and end-to-end anastomosis, or excision and vessel grafting, may re-establish the main arterial flow.

Subfascial (Arterial) Hematoma.—Injuries to small arteries, or small wounds in large arteries, may cause a hemorrhage that is confined to subfascial compartments, which, by compression, act as a tourniquet. Complete venous obstruction and nearly complete arterial obstruction may result even though the arterial segments are open.

FIG. 147.—Pathologic fracture through shaft of the femur. The bone has been destroyed by metastatic cancer from the thyroid.

tion must be suspected when a fracture results from slight trauma or when it is relatively painless.

The common conditions associated with pathologic fractures are:

A. *Systemic diseases:*
   1. Osteogenesis imperfecta
   2. Paget's disease of bone
   3. Hyperparathyroidism (osteitis fibrosis cystica)
   4. Tabes dorsalis
   5. Osteoporosis

associated with vascular insufficiency due to laceration of the artery, thrombosis, persistent spasm or subfascial hematomas, which require appropriate operative measures. The best guarantee against Volkmann's ischemic contracture is (*a*) early, gentle and accurate reduction of the fracture and (*b*) careful attention to the position of the elbow and cast to see that the circulation is not compromised.

Ischemic contractures may also occur in areas other than forearm and hand. For example, they are occasionally encountered in the leg and foot after supracondylar fractures of the femur, and rarely in the hand following forearm fractures or in the foot following leg fractures. The measures described for the prevention of ischemic contracture following supracondylar fracture of the humerus are equally important in other areas.

Nerve Paralysis.—The surgeon who undertakes the treatment of any patient for a fracture must carefully search for associated nerve injuries and make a written record of his findings. If he omits this detail, he does the patient a great disservice and leaves himself liable to a malpractice suit.

In an occasional instance, the presence of nerve deficit may indicate the need for early open operation in order that the nerve may be examined and repaired. An early or initial nerve deficit may result from laceration, stretching or contusion. Nerve palsy appearing later may result from manipulation or open operation or from nerve compression by hematoma. Still later, nerve paralysis may result from pressure caused by the cast or traction apparatus or by incorporation of the nerve in callus. The radial nerve is particularly liable to injury because of its intimate relationship with the midportion of the shaft of the humerus. The median or ulnar nerves may be injured in fractures in the elbow region. In shoulder dislocations the axillary nerve may be stretched. Hip dislocations may cause injury to the sciatic nerve. The pressure of a plaster cast over the upper fibula may result in foot drop due to common peroneal nerve damage.

The surgeon must have a working knowledge of the anatomy of the peripheral nerves and must be familiar with simple reliable tests for their motor and sensory function. Furthermore, he is obligated to examine the patient periodically for evidence of altered nerve function during the period of fracture healing and to record his findings on the clinical record.

It is evident that all patients with injuries to the spine should be examined for the presence of spinal cord injury. Most patients with

Circulatory insufficiency distal to the injury may be so severe as to result in loss of the extremity. Relief of arterial and venous compression can be provided by prompt wide incision of the fascia, allowing the deeper tissues to bulge outward. Blood clots should be evacuated, and bleeding controlled by ligature or vessel suture.

VOLKMANN'S ISCHEMIC CONTRACTURE.—Volkmann (1881) described this disorder and attributed it to circulatory insufficiency. He likened the hardening of muscles to rigor mortis and differentiated the condition from paralysis due to nerve deficit. He implicated tight bandages, tourniquets, and lacerations and contusions of the large arteries as causative factors. Usually Volkmann's contracture occurs in the upper extremity, and most often in association with supracondylar fracture of the humerus, "the paramount fracture of childhood" (Akin). It is necessary for the surgeon to be acquainted with the cause and prevention of this tragic deformity.

Whether ischemic contracture is the result of arterial insufficiency, venous obstruction, or both, makes little difference from a practical standpoint. It may be prevented in almost all instances by prompt, gentle and accurate manipulative reduction of the fracture and, after reduction, by seeing to it that neither the position of immobilization, the cast nor the bandages cause circulatory impairment. This fracture represents a real emergency. Treatment should be instituted within four hours and preferably within two hours after injury. In the usual supracondylar fracture of the humerus, the distal fragment is displaced dorsally or posteriorly, and manipulation consists first of strong traction to overcome shortening (this relieves any pressure on the brachial artery by the proximal fragment of the humeral shaft) and then flexion of the elbow, combined with forward traction to reestablish the "lower humeral angle" of about 40 degrees. Clumsy manipulations or flexion of the elbow without sufficient longitudinal traction may compress or lacerate the brachial artery.

After the careful reduction and positioning of the fragments, the degree of flexion at the elbow must not be so great as to cause pallor, cyanosis, coldness of the hand or disappearance of the radial pulse. Furthermore, the status of the circulation in the extremity must be checked frequently during the subsequent forty-eight hours. Failure to obtain reduction, or persistent evidence of circulatory deficiency following reduction, should cause the surgeon to abandon manipulative reduction in favor of skeletal traction.

In rare instances, supracondylar fractures of the humerus may be

force which caused the pelvic fracture. Rupture of the distended bladder may occur *in either its intra- or its extraperitoneal portions.* In either case, the patient does not void any sizable amount of clear urine. Although a catheter may be passed easily into the bladder, *only blood or small amounts of blood-stained urine* will be obtained. In questionable cases, x-ray examination after instillation of a sodium iodide solution will reveal extravasation from the bladder.

Fractures of the *ischiopubic rami may lacerate or completely sever* the membranous urethra. The patient ordinarily cannot void, and the urinary bladder distends. He wants to void and calls his doctor's attention to *his inability* to pass *urine. A few drops of blood may* be evident at the urethral meatus. Attempts to pass a catheter fail, and only blood is obtained. Treatment consists of suprapubic cystostomy, an indwelling urethral catheter and, *in some situations,* repair of the laceration.

Rupture of the spleen, liver, kidney, pancreas and hollow organs of the abdomen and thorax may also occur (See Chapter 23, Abdominal Injuries, and Chapter 31, Chest Injuries).

Muscle Atrophy.—Muscle atrophy occurs rapidly if the extremity is immobilized and the muscles are not used. Regular exercise of such muscles as can be contracted within the limits of the type of immobilization used is important. Without disregarding the rule to "immobilize the joint above and the joint below the fracture," no more immobilization than necessary should be instituted. Casts which include the hand should leave the fingers and thumb free to move. If a finger is immobilized for fracture, only that finger is immobilized. The cast for a Colles' fracture should extend only to the distal flexion crease of the palm, in order that the fingers may be fully flexed and extended. The thumb should be left free to move. The fingers and toes must be exercised regularly to prevent joint stiffness and muscle atrophy. When immobilized, the quadriceps femoris and thigh adductor muscles are prone to undergo rapid atrophy. The quadriceps group can be exercised by moving the patella ("pulling it up") within the cast without bending the knee. These exercises can be learned easily by the co-operative patient. Finger and toe exercises, where appropriate, should be carried out for five minutes of each hour by the clock during the patient's waking hours. The surgeon who reduces and immobilizes a fracture has not done a good job until he instructs the patient in the appropriate exercises and sees to it that they are carried out.

fractures of vertebrae and with "cord signs" do not benefit from laminectomy. Patients who show little evidence of cord damage initially will sometimes exhibit signs of increasing cord compression. Under these circumstances, laminectomy may be indicated. Sometimes a depressed vertebral lamina may be elevated with benefit to the patient. Usually, however, the cord will have been severely damaged at the

FIG 148.—Fractures of the pelvis with rupture of the bladder in a male

original injury, and at operation it will be found to be soft and gelatinous ("pulpified") Little or no recovery of function can then be expected.

MUSCLE AND TENDON DISRUPTION —In unusual circumstances, fractures may be accompanied by the disruption of muscle or tendon. Primary repair in closed fractures or delayed repair in open fractures is indicated in rare instances Thus, a fracture of the inferior portion of the patella may be disabling largely because of disruption of the patellar tendon. Rarely, in supracondylar fractures of the femur the quadriceps muscle may be severed at its musculotendinous zone.

VISCERAL COMPLICATIONS.—Pelvic fractures may be associated with rupture of the urinary bladder (Fig 148) This may be due to a bony spicule which lacerates the bladder extraperitoneally or to the direct

force which caused the pelvic fracture. Rupture of the distended bladder may occur in either its intra- or its extraperitoneal portions. In either case, the patient does not void any sizable amount of clear urine. Although a catheter may be passed easily into the bladder, only blood or small amounts of blood-stained urine will be obtained. *In questionable cases, x-ray examination after instillation of a sodium iodide solution will reveal extravasation from the bladder.*

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Myositis Ossificans.—Myositis ossificans consists of bone formation beyond the fracture in muscle, fascia or joint capsule. It most commonly occurs in the anterior elbow region after supracondylar fractures of the humerus. It may develop from stripped periosteum or from the fracture hematoma, or in the joint capsule. Conservative treatment is indicated. Active motion within the limits of pain is permitted, but forceful passive motion must be avoided. Ordinarily, this aberrant new bone gradually undergoes absorption. Operative removal of bony spurs which limit motion should be delayed for many months after the original injury because of the dangers of extending the ossification.

Delayed Ulnar Palsy.—This condition may occur after elbow fractures which result in a cubitus valgus (increased elbow carrying angle) deformity. In most instances, it results from a childhood fracture of the lateral humeral condyle leading to epiphyseal injury, arrested growth and severe cubitus valgus deformity. The stretched ulnar nerve "decompensates," and paralysis results. The treatment is transposition of the ulnar nerve from the posterior to the volar aspect of the elbow. Tension is thereby relieved, and restoration of function may be anticipated.

Postcast Edema.—Edema of the foot and leg commonly occurs *after removal of a supporting long leg cast. If allowed to persist,* the leg may become permanently swollen, indurated and possibly ulcerated. Edema may be minimized by elevation, massage and supporting elastic bandages whenever the patient is upright. If edema is prevented early, late chronic disability may be avoided.

FRACTURE APHORISMS

(From *Outline of the Treatment of Fractures,* by American College of Surgeons, 1954.)

1. Treat every case of injury as a fracture until it is proven to be otherwise. Protect and immobilize all injured patients until the diagnosis is made. "Splint 'em where they lie."
2. Always use gentleness and care in handling any broken limb. Roughness is inexcusable.
3. Use only the simplest methods of examination.
4. Eliminate all unnecessary handling of the injured part.
5. Never deliberately search for crepitus.
6. Disturb the patient as little as possible.

7. Do not be deceived by the absence of deformity and disability; *in many cases of fracture some ability to use the limb persists.*
8. Make sure that you are not dealing with more than one fracture.
9. See that the patient has an early suitable x-ray examination.
10. Examine for nerve lesions and for associated injuries before attempting reduction.
11. Active movements affect muscles and mind, *usually* strengthening both.
12. Apply slow, steady pull to relax the muscles and not to produce irritation.
13. Bring the fragment which can be controlled into alignment with the fragment which cannot. (Usually distal to proximal.)
14. Watch the circulation distal to the injury.
15. A small fracture may mean a large disability.
16. *Reduce the fracture with as little delay as possible. Do not wait* for the swelling to go down.
17. No splint is used to reduce a fracture; a splint is intended to maintain reduction.
18. Splints made to fit everybody rarely fit anybody.
19. Early, active movements help healing, if the fracture is not disturbed.
20. *Continued pain usually indicates incomplete reduction or improper* splinting.
21. Make certain that traction is checked hourly.
22. Shoulders frequently freeze unless they are moved early and often following fracture of the forearm or wrist.
23. The measure of successful treatment is the usefulness of the limb as a whole.
24. *Compare the well with the injured part.* X-rays of both extremities on one plate, with a single exposure, are often helpful.

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CHAPTER 30

Head Injuries

IN CIVIL LIFE, craniocerebral trauma is not exceeded in frequency or severity by that met among military personnel during active warfare. The factors that appear to influence the growing numbers of head-injured patients include: the toll of the road, the hazards of the machinery of modern industry and agriculture, competitive sports involving bodily contact, thrill-charged recreations, alcoholism and—pervading our over-all activity—a growing indifference to, and abrogation of, responsibility for the welfare of others.

Among unselected cases, such as are admitted each year to large metropolitan hospitals, *slightly more than 70 per cent of the patients* recover regardless of the treatment employed, and between 8 and 12 per cent die in consequence of the overwhelming character of the trauma. In the remaining patients (15–20 per cent), the moderately to severely injured, mortality and morbidity depend closely on the early *institution of appropriate therapeutic and preventive measures.* Most of these patients require only such conservative measures as are available in every modern hospital.

Only 4–5 per cent of the unselected cases can be significantly helped by open operation during the first week or so following trauma. This does not mean that, under conditions of practice, only 4–5 per cent of head-injured patients undergo operation, but that not more than this number can be justly asserted to receive benefit. Unfortunately, some surgical efforts prove futile, some turn out to be unnecessary, and others are capable of relieving only a small part of the total damage present.

The foregoing data carry an encouraging message to the physician upon whom responsibility for the management of head-injured patients devolves. They indicate that in some 80 per cent of patients the clinical

outcome is largely predetermined, independent of his skill, and that in an additional 15 per cent the patient's survival and the minimizing of residual neurological and psychologic deficits can be achieved through the intelligent exercise of certain orienting principles and specific conservative measures which are essentially the same, from case to case, regardless of the nature of the intracranial pathologic process. Moreover, the utilization of these measures requires no very detailed knowledge of neuroanatomy, pathogenesis or pathology; and the measures can, with practice, be as effectively applied by the skilled general practitioner as by the neurological specialist. Lastly, the criteria indicating the necessity for early surgical inquiry and intervention do not depend on a precise diagnosis but on signs that can be almost as readily observed by the layman as by the highly trained clinician. Hence, the only circumstance in which the general practitioner stands at a serious disadvantage, as compared with the neurosurgeon, is that in which open surgery is required.

Ideally, all cases requiring operation should be referred to a neurosurgeon or a general surgeon experienced in the relatively simple neurosurgical technics required. But if neither of these is within easy summons, an advantage still rests with the general practitioner; for, with the exception of cases exhibiting rapid deterioration (e.g., epidural hematoma), the margin of safety of the head-injured patient suspected—early enough—of being in need of operation is usually sufficient to permit his being transported to a suitable clinic. Military experience in World War II and in the Korean action amply demonstrated that it is better to send a patient to a competent surgeon whose operating and nursing teams are at close hand than to attempt to manage him under handicaps prevailing locally for fear of the dangers of transportation.

Meanwhile, up to the moment of transportation and during the trip itself the very same conservative measures called for in nonoperative cases are indicated. (It may be added that they are likewise required *after* open surgical measures have been executed.)

All this indicates that the majority of head-injured cases falling under the care of general practitioners and surgeons may remain there. There is little substance to that psychologic barrier against accepting responsibility for the head-injured which prevails so generally among practitioners and which so often results in disservice to patients by default.

Basic Aims of Treatment of Head Injuries

What, then, are the realizable primary goals toward which the clinician should direct his efforts? They are simple and few: (1) to reduce mortality and morbidity to the acceptable minimum cited above; (2) to provide every patient with the time-tried, effective methods of treatment now available; and (3) to so conduct matters that few of the patients who are unable to profit from operation will be subjected to operation and no patient harboring a clinically significant, surgically amenable, lesion will be deprived of the benefits of operation.

It will be noted that other frequently cited goals, such as (*a*) comprehension of the pathologic and pathogenetic processes that may obtain in the head-injured patient and (*b*) the making of precise diagnoses, have been omitted above. The reason for the omission is that they represent *secondary*, rather than *primary*, goals; i.e., they are means to ends, not ends in themselves. Moreover, as we shall shortly see, they are in no sense indispensable prerequisites to effective activity in behalf of the patient. To assume that they are is to set up crippling psychologic barriers that deter the physician from instituting such valuable services as he is capable of bringing the head-injured patient. This point of view requires further elucidation.

Orthodox medical teaching emphasizes the desirability of proceeding in orderly sequence from the incidence, etiology and pathology of "disease" to pathogenesis, signs and symptoms, syndromes, diagnosis and treatment This sequence should be recognized distinctly as a scientific ideal. Where realizable, it has paid welcome dividends in medical practice, and for this reason deserves to be honored. Unfortunately, however, there are many morbid conditions in medicine in respect to which the present insistence upon the ideal proves inept. Most cases of craniocerebral trauma belong in this category, for neither the general practitioner nor the neurological specialist can be certain in a given case that he comprehends precisely the prevailing pathogenetic and pathologic factors and the dynamic changes they undergo from one hour to the next. The best the physician can do is to draw a "reasonable" diagnostic inference, i e., make a good "guided guess" and act accordingly, remaining ever aware of the fallibility of his inferences.

If the matter of correct diagnosis were the major issue at stake,

the realistic circumstances referred to above would bode ill for both patient and doctor. But despite them, our primary goals can be, and in many hospitals regularly are, approximated year in and year out. Granted that it constitutes a desirable state of affairs in medical problems at large to know the etiology, pathology and pathogenesis of the disorders prevailing in each patient, the lack of such knowledge does not necessarily constitute a serious deterrent to effective therapy. It is not essential to know the cause and origin of a fire to put it out.

Orienting Principles in the Approach to the Problems of the Head-Injured Patient

We will now consider four broad principles that appear to have paid dividends in dealing with the various problems of the head-injured patient:

1. *Neurological signs and symptoms result from disturbances in neurohumorophysiologic mechanisms and are clinically alike, regardless of the etiologic and pathologic agents that produce them.* In less abstract form, this indicates that, for example, hemiplegia due to an epidural clot is indistinguishable on clinical grounds from that due to (*a*) cortical contusion and laceration, (*b*) encephalomalacia secondary to traumatic thrombosis of cerebral vessels, (*c*) pulpification of a lobe of the brain, (*d*) cerebral edema, (*e*) multiple petechial hemorrhages and/or (*f*) a shift of the brain stem across the midline. What holds true for hemiplegia holds for most other neurological signs and symptoms. Hence, no amount of delineation of signs and symptoms per se can furnish the physician with *confident* knowledge regarding the pathologic agent(s) at work in a particular patient. For this reason, the clinician is obliged to deal with his patient in terms of *probabilities* rather than of certainties. In so doing, he may be—and not infrequently proves to be—in error.

2. *The pathologic processes that exist in the traumatized brain cannot always be described in terms of the gross and microscopic findings* Our conventional pathologic technics are capable of disclosing only a limited number and kind of pathologic changes. They fail to reveal many subtle processes that modern microchemistry and biophysics have begun to identify—e.g., the cytologic effects of various degrees of ischemia, hypoxia, hypoglycemia, retention of catabolites, escape of acetylcholine, intra- and extracellular fluid and electrolyte imbalance, positive and negative "voltage drifts," acid shift of pH,

depolarization and other surface-tension alterations of boundary-phase membranes, extinction, dysfunction of respiratory pigments and other organic enzyme systems, derangements of colloid dispersions, reversions of aperiodic to periodic crystals, etc. There is reason to suppose that such subtle changes as those just mentioned are in many instances sufficient to produce serious morbidity—even death—*with* or *without* the coexistence of lesions revealed by gross and/or microscopic examinations. If this is a tenable view, the more obvious lesions in some cases may be responsible for only part of the mortality and morbidity of the head-injured patients and in other cases may be largely incidental, so that measures directed at their correction not only prove ineffectual but actually add to the stress under which the patient labors.

3. Among the seriously injured, *multiple pathologic processes at multiple sites* (as contrasted with unit-factor lesions) *are more frequently found than not.* Thus, a patient seriously enough injured to develop an epidural clot frequently also sustains contusions of the brain, venous bleeding into the subdural space, multiple petechial hemorrhages scattered throughout the white matter, a gross intracerebral clot and/or circumferential cerebral edema. As a consequence, the classical "syndrome of the epidural clot" can easily be masked by clinical signs engendered from coexisting lesions and thus fail to be clinically detected. Accordingly, the experienced physician recognizes that the *absence* of a given syndrome provides no assurance that the pathologic lesion to which that syndrome is conventionally imputed is absent, also, that its *presence* in no way guarantees that the lesion conventionally imputed to it will be found at operation or post mortem, much less that it constitutes the main "cause" of the patient's difficulties.

4. Our final orienting principle derives from the first three and indicates that *it is necessary to adopt a philosophy of clinical agnosticism.* The experienced physician knows that he cannot be sure of his inferences regarding a particular case and is careful not to repose false confidence in them. He renounces the elementalistic rules-of-thumb that spring from confident certainty in diagnosis and remains continually alert to the ever changing picture presented by the patient, guided by probabilities rather than complacent certainties. He actively seeks evidence which controverts his tentative diagnostic and therapeutic hypotheses and holds himself ready to revise or abandon first inferences in favor of more tenable hypotheses.

the realistic circumstances referred to above would bode ill for both patient and doctor. But despite them, our primary goals can be, and in many hospitals regularly are, approximated year in and year out. Granted that it constitutes a desirable state of affairs in medical problems at large to know the etiology, pathology and pathogenesis of the disorders prevailing in each patient, the lack of such knowledge does not necessarily constitute a serious deterrent to effective therapy. It is not essential to know the cause and origin of a fire to put it out.

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If the cranium and its contents actually constituted a rigidly closed physical system, such an increase of pressure would be felt equally throughout all parts of the cranial cavity. However, the skull does not amount strictly to a closed system. In the infant with patent fontanels, in the young child with cranial sutures capable of considerable spreading (dehiscence) and in the adult with foramens of various sizes at the base of the skull, the condition of physical rigidity is not met. Hence, the theoretically expected equality of hydrostatic phenomena does not obtain. Again, the fact that the brain is more securely fixed to the floor of the skull than elsewhere determines certain departures from the simple hydrostatic scheme. In addition, three partly separate intracranial compartments exist—viz., the two formed by the falx cerebri and a third by the tentorium cerebelli. Finally, measurable differences in strain, stress and torque factors exerted on vulnerable cerebral vessels and inequalities of circulation at various places within the cranial cavity result in local pathologic responses, and therefore in local disparities in intracranial pressure.

Cerebrospinal Fluid.—The cerebrospinal fluid is a clear, protein-poor, watery liquid of high electrolytic concentration, usually regarded as a virtual dialysate of plasma in hydrostatic and osmotic equilibrium with blood. Until the late nineteen thirties, it was believed to be formed entirely in or by the choroid plexus. But evidence now available suggests that the major part of the fluid is formed within the substance of the brain, whence it empties into the ventricles and subarachnoid field via the perineuronal and perivascular (Virchow-Robin) spaces.

It is presumed that the gradient of the cerebrospinal fluid pressure falls off progressively from the intraventricular to the subarachnoid pools; and hence, that under normal conditions the fluid leaves the ventricles and iter of Sylvius, passes through the foramens of Magendie and Luschka and reaches the subarachnoid space of the *cisterna magna* in the posterior fossa. From there it has direct access to the subarachnoid space of the spinal canal, which, in the healthy adult, contains about 70 ml. of cerebrospinal fluid. The fluid also has indirect access from the cisterna magna to the subarachnoid spaces of the cerebral hemispheres. Eventually it reaches the arachnoid villi of the hemispheral convexities, through the endothelial membranes of which it flows into the venous blood stream of the dural sinuses. A certain fraction of the fluid is said to be steadily absorbed via lymphatic channels along the peripheral nerves.

The quantity of "free" (readily drainable) intracranial cerebro-

The foregoing is not meant to imply that the clinician need concern himself only with the implementation of superficially empirical measures, vouchsafing basic scientific and medical knowledge and discounting clinical syndromes as being fatuous, when not actually misleading Rather, it is meant to give assurance to *otherwise* experienced physicians that they need not mistrust their abilities to perform creditably when confronted with the majority of head-injured patients.

PATHOPHYSIOLOGIC CONSIDERATIONS IN HEAD INJURIES

In recent years, a number of observations—e.g., knowledge of the principles known as "the Monroe-Kellie doctrine and of the mechanics of the cerebrospinal fluid and of cerebral concussion—have helped to clarify the pathophysiology of craniocerebral injury and to render the clinical phenomena more understandable.

MONROE-KELLIE DOCTRINE.—This doctrine may be summarized as follows: The cranium is a relatively closed, rigid vault, the total contents of which (brain substance, cerebrospinal fluid and blood) must remain approximately the same at all times. A change in volume of one or more of these factors must promptly be compensated for by a reciprocal change in volume of one or more of the other factors. Hence, any agent (e.g., a blood clot) which usurps appreciable space necessitates a compensatory partial displacement of one or more of the agents normally present. The cerebrospinal fluid is most easily displaced and therefore yields first, after which the circulating minute blood volume of the brain begins to suffer. When the blood volume falls below a critical level, cerebral ischemia, hypoxia, hypoglycemia and hypercapnia supervene and lead to the overt appearance of clinical signs and symptoms—systemic, neurological and psychologic. The least displaceable agent is the brain tissue itself. The medulla and tonsils of the cerebellum can, to a very limited degree, be herniated through the foramen magnum; but in the acute case, this constitutes a grave complication and is, of course, incapable of effectively meeting the physiologic problem. A downward displacement of the hippocampi uncus of the temporal lobe below the incisura of the tentorium cerebelli may also occur; but, like medullary herniation, this displacement is ineffectual as a mechanism of compensatory displacement.

As the intracranial space is progressively encroached upon by a blood clot or similarly acting foreign agent, the limits of displacement of the agents normally present are ultimately reached. Intracranial pressure then begins to rise.

CEREBRAL CONCUSSION.—*The mechanical force of a physical agent impinging on the head is rapidly dissipated and follows lines of variable resistance, depending on the site and direction of the force, the shape of the skull, its resiliency, suture lines, bony buttresses, etc.* A considerable fraction of the initial force is transmitted to the cerebrospinal fluid, which, being largely incompressible, imparts much of its mechanical energy to the neurons it bathes. The result is a generalized, fairly simultaneous discharge of energies stored within the "resting" neurons; i.e., a synchronous "depolarization" of cell membranes occurs. There ensues an intense, but short-lived, excitation of the muscles and glands upon which the neurons so depolarized ultimately play. Immediately following this discharge, a period of "extinction" (abolition) of neural function supervenes. The clinical counterparts of these "excitatory" and "paralytic" phases of cerebral concussion consist of a brief tonic spasm of the bodily musculature, alterations of respiratory and cardiovascular functions, temporary impairment of consciousness and loss of corneal reflexes. Mild confusion, dizziness and headache usually ensue for a variable period up to several hours after the paralytic phase passes.

In mild to moderate concussion of the brain, gross pathologic changes are rarely demonstrable, and microscopic changes, when detectable at all, are usually inconspicuous. Windle states that on occasion pathologic changes appear as early as 30 seconds after the physical violence and that they commonly reach their maximum at the end of the first week, thereafter regressing rapidly. The most characteristic histologic feature of such changes is chromatolysis. In the more severe degrees of concussion, less readily reversible degrees of intracellular and molecular derangements are produced. Focal and/or diffuse microscopic hemorrhages may be demonstrable within and upon the surface of the brain. These may be serious enough to interfere with the circulation of blood and the egress of cerebrospinal fluid. The resulting deficiency of cellular respiration and the perverted response of cells to ischemia, hypoxia, hypoglycemia and *hypercapnia* lead to the development of edema. The latter mechanically compromises the smaller vessels of neighboring tissues and induces more ischemia, hypoxia, etc. A vicious circle is thus established and leads eventually to cellular death.

Encephalomalacia may develop so rapidly following severe injury that a previously normal ventricular system may show a perceptible degree of dilatation within a week. Such dilatation may continue to

spinal fluid amounts to approximately 50 ml. An additional quantity occupies the intracerebral perivascular and perineuronal spaces.

Normally, the cerebrospinal fluid pressure varies from 80 to 180 mm. H_2O (6–13 mm. Hg). Since, level for level, the hydrostatic pressures of a fluid in a relatively closed system are nearly the same, and since the fluid-containing spaces of the central nervous system are normally in free communication with one another, the pressure of the cerebrospinal fluid will be found similar at whatever points the system is tapped. Hence, if the patient lies in the lateral decubitus position, the pressures of the cerebrospinal fluid in the ventricular, cisternal and lumbar subarachnoid spaces will be virtually the same, *provided no spinal subarachnoid block exists.*

Alterations in cerebrospinal fluid pressure closely parallel changes in systemic and intracranial venous pressure. Whenever the subject strains, coughs or sneezes, the increase of intrathoracic and intra-abdominal pressures is dissipated in part along the venous channels of the brain and spinal cord. This interferes with venous drainage from the head and results in momentary increase in blood volume of the brain and a compensatory displacement of intracranial cerebrospinal fluid. For similar reasons, the forced Valsalva maneuver (closing the nose and mouth and "straining") raises the pressure of cerebrospinal fluid. The Queckenstedt test, in which the jugular veins on each side of the neck are simultaneously compressed for ten seconds while lumbar spinal manometry is in process, depends on similar physiologic principles.

In head-injured patients the Queckenstedt test is contraindicated. Its sole clinical usefulness is in gaining information concerning the patency of the spinal subarachnoid space. It is dangerous because it may precipitate an active intracranial hemorrhage from damaged vessels which might hold but for the added stress of jugular vein occlusion

Although severe rises of intracranial cerebrospinal fluid pressure provoke a rise of arterial blood pressure, the reverse does not hold. Arterial hypertension, even of severe degree, has no measurable effect on cerebrospinal fluid pressure There is one exception to this statement: When circulatory failure ensues and normal capillary resistance gives way, allowing arteriolar pressures to be mediated across the vascular tree to the venules and veins, venous pressure rises and produces the conditions described above, resulting in an elevation of the cerebrospinal fluid pressure.

C. Ecchymoses and edema
   1. General: of scalp, facial and neck tissues
   2. Special
      a) Periorbital (if delayed, this suggests fracture of the frontal bones, base and/or vault)
      b) Postauricular (if delayed, this ["Battle's sign"] constitutes strong, presumptive evidence of fracture of the petrotemporal bone)

D. Subcutaneous emphysema of—
   1. Forehead (with fracture into frontal sinus)
   2. Cheek and nose (with fracture into antrum)
   3. Neck (with tracheal fracture and lung puncture)

E. Hemorrhage *
   1. Free
      a) Lacerations and avulsions
      b) Epistaxis
      c) Pharyngeal
      d) Middle ear

      (b–d) All suggest basilar fracture. If blood is mixed with cerebrospinal fluid, it constitutes conclusive evidence thereof.
   2. Trapped
      a) Subaponeurotic (cephalohematoma)
      b) Intraocular
      c) Retinal (with or without detachment)
      d) Retropharyngeal (suggests cervical fracture-dislocation)
      e) Tympanic cavity (without rupture of drumhead)

F. Imbedded foreign bodies: dirt, gravel, cinders, twigs, fragments of glass, stone, wood, metal, pieces of hat, etc.

G. Loosening, fractures and avulsion of teeth

II. *Injuries to Bones of Skull, Face and Neck*

   A. Fractures of skull (simple and compound) located in the vault, the base or both

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major, responsible factor at work. Obviously, syncope arising from many causes—diabetes, hyperinsulinism, apoplexy, cardiac catastrophe, drug intoxication, etc.,—may be complicated by a fall, resulting in external marks of violence without significant cerebral counterpart On the other hand, moderate to serious cerebral trauma secondary to a fall may add to, and even supersede in severity, the original cause of syncope. Wariness will serve the clinician well in such instances.

* Orificial bleeding should immediately suggest the possibility of fracture of the base of the skull compounded through the paranasal sinuses, middle ear or mastoid cells. Epistaxis and pharyngeal bleeding suggest basilar fracture compounded [illegible]

[illegible] through the tympanum but can be recognized through a bulging, bluish red drumhead. *The orificial discharge of cerebrospinal fluid or of cerebrospinal fluid mixed with blood permits a positive diagnosis of basilar fracture, irrespective of the negative results of routine x-ray examination.*

progress for several months after clinical recovery has apparently occurred

In an important study, Denny-Brown and Russell (1941) drew attention to the disparity between the damage sustained by the skull and that of its contents, depending on whether the head is or is not free to move at the moment of physical violence. They showed that, when the head of the experimental animal is fixed, the skull may be severely damaged, while the brain suffers minor injury only. There may be no loss of consciousness. When, on the other hand, the freely moving head is injured, the skull may wholly escape injury, while the inertia of the brain results in changes in velocity around an infinite number of axes (torque forces) as well as changes in line with the forces of acceleration and/or deceleration. The brain slaps against the irregular nubbins of the inner table of the skull and the edges of the falx and tentorium. Thus, contusions, lacerations and, in severe cases, gross pulpification of brain tissues may be produced. Arteries and veins may be torn, and gross microscopic hemorrhages may develop in the epidural, subdural, subarachnoid, subpial, intracerebral and intraventricular regions.

Gurdjian and Lissner (1944) concluded that, because of the imperfection of the spherical shape of the head, the inequalities of its thickness and elasticity, the weaknesses produced at the base by numerous foramens and the variable strength of bony reinforcements and individual differences, it is not at present possible to calculate mathematically or predict accurately the fracture patterns of the human skull.

Pathologic Findings in Head Injuries

The manifest and hidden pathologic findings most commonly encountered in craniocerebral trauma may be classified into three groups, as follows:

I. *Marks of Violence on Scalp, Face, Neck and Teeth* *
   A. Abrasions (including "brush burns") and contusions
   B. Lacerations and avulsions

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*Sometimes the physician is required to attend comatose or confused patients lacking a satisfactory history. Such cases raise the problem of the differential diagnosis of coma,
The absence o .
cerebral trauma, [illegible]
the examiner into supposing that craniocerebral trauma is the sole, or even the

2. Focal
3. Circumferentially spreading
4. "Persistent" type of Trotter

F. Intracellular, intramolecular and other submicroscopic derangements

Clinical Symptoms and Signs in Head Injuries

The clinical signs and symptoms which frequently appear in head-injured patients may be classified under three general headings—(I) Modifications of the "State of Consciousness," (II) Vegetative Disturbances and (III) Somatic Neurological Disturbances—as follows:

I. *Modifications of the "State of Consciousness"* *

A. *Psychomotor and emotional activities*
1. Mania, thrashing and shouting behavior
2. Actively resistive, abusive, negativistic and combative behavior
3. Hypomania and delirium
4. Restless, insomnic, voluble behavior
5. Normal behavior
6. Apathy, inaccessibility
7. Bradykinetic, random and semipurposive movements
8. Somnolence, stupor
9. Comatose and preagonal states; coma vigil

B. Intellectual and social activities
1. Confusion, disorientation, dulled perception
2. Inappropriate behavior
3. Aphasia
4. Amnesia
   a) Anterograde (for events following injury)
   b) Retrograde (for events just preceding injury)
5. Distractibility, inattentiveness
6. Repetitiveness, perseveration
7. Incontinence of stool and urine

II. *Vegetative Disturbances*

A. Blood pressure
1. Normal, increased, decreased; instabile
2. Normal; bradycardic, tachycardiac; instabile; bounding, thready; coupled

B. Respiratory rate, depth and character
1. Early: stertorous (slow, deep and noisy)
2. Late rapid, shallow; inspiratory phase shorter than expiratory, mucus present or absent; hiccoughing

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*Diurnal and even hourly variations between the extremes of responsiveness are not infrequently observed. *Wide swings should be regarded as evidences of grave insult.*

1. Linear
2. Penetrating
3. Perforating
4. Comminuted
5. Stellar, bursting
6. Depressed (linear, comminuted, pond, gutter and disk types)
7. Dehiscence of sutures

B. Fractures and/or dislocations of facial bones, simple and compound

C. Fractures and/or dislocations of cervical spine (usually simple and at C5–6), with or without spinal cord dysfunction *

1. Compression ("wedging") of vertebral body
2. Facets, pedicles, laminae, odontoid process
3. Anterior or posterior subluxation with ligamental tears

III. *Injuries to Intracranial Contents*

(Brain, meninges, blood and blood vessels, cerebrospinal fluid and its cisterns, ventricles and channels)

A. Concussion (commotio cerebri)

B. Laceration and contusion (direct and contrecoup types)

1. Dura mater and/or venous sinuses
2. Pia-arachnoid
3. Cortex (cerebral and/or cerebellar)
4. Brain stem

C. Pulpification of lobe (usually temporal or frontal)

D. Hemorrhage

1. Epidural (from anterior and middle meningeal arteries)
2. Subdural (commonly of venous origin, but may be from smaller arteries or arterioles and lacerated cisterns)
3. Intracerebral
4. Intraventricular
5. Extravasations, punctate and interstitial hemorrhages, diffuse or focal (of magnitude not amenable to surgical removal)

E. Brain edema

1. Diffuse

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*The association of injuries of the cervical spine and cord with head injuries and the frequency with which they are overlooked require special comment. Fracture-dislocations of the cervical vertebrae and, in less severe cases, traumatic myositis and ligamentitis are frequently produced when physical violence is de-

of mere meningeal irritation due to subarachnoid bleeding; and the neurological consequences of concussion, edema and/or hemorrhage of the cervical cord have often been erroneously imputed to the concomitant brain injury.

On occasion, a cervical dislocation produced by a frontally delivered impact or by a whiplash injury, in which the inertia of the head results in a snap and rebound when the body is suddenly accelerated from behind, may spontaneously reposition itself, yet leave serious neurological deficits

phenomena in the same case; and fluctuations are to be expected from hour to hour, depending on variations in respiratory and circulatory functions, the ebb and flow of cerebral edema and submicroscopic derangements in the cellular and extracellular compartments.

Clinicopathologic Syndromes in Head Injuries

Up to this point, attention has been given to the pathologic processes and the clinical signs and symptoms commonly encountered in cases of craniocerebral injury. We shall now review and evaluate the attempts on the part of students of the past to discover a regular correspondence between (*a*) particular pathologic lesions and (*b*) the sequential course and combination of signs and symptoms said to constitute "syndromes." The ultimate purpose of such attempts has been to enable the clinician to diagnose the former through demonstration of the latter.

Unfortunately, the traumatic syndromes have been overexploited in many textbooks; i.e., insufficient stress has been laid on the fact that they are most useful in the presence of "pure" (unit-factor) lesions. As was noted above, multiple lesions are by far the most prevalent in head trauma, and the clinical features of one lesion tend to be masked by those of other lesions. This should not be taken to mean that the clinician encountering signs manifestly consistent with a given traumatic syndrome needs not *suspect* the lesion conventionally associated therewith; it merely warns that he should continue to proceed in terms of probabilities, anticipating the possibilities that (*a*) the suspected lesion will not be found at all, (*b*) other lesions may prove wholly responsible for the clinical picture, and (*c*) if the suspected lesion is found, other lesions, some surgically amenable, some not, may coexist and require attention.

In this connection, the familiar *syndromes of epidural hemorrhage and increased intracranial pressure* will now be examined:

The essential features of the former (epidural hemorrhage) consist of (*a*) unconsciousness immediately following injury and prevailing for a varying period (commonly three to twenty minutes), (*b*) next a lucid interval; and (*c*) then a gradual development within one to several hours (or days) of: unilateral headache, somnolence, stupor and coma; enlargement of one pupil; conjugate deviation of the eyes toward the side of the clot, paresis and increasing spasticity with or without seizures of the contralateral limbs, vomiting; rise in blood

C. Temperature. hyperthermia, average 101°–104° F.; if uncombated, may reach 107°–109° F.
D. Upper gastrointestinal tract: nausea, vomiting, regurgitation of bile
E. Skin
   1. Anhidrosis (except when the brain injury is complicated by toxic or infectious processes)
   2. Flushed or cyanotic head, neck and chest; preagonal mottling of extremities
   3. Goose flesh in decerebrate states and other severe injuries
F. Sugar metabolism: frequently hyperglycemia and glycosuria
G. Sphincters: incontinence of stool and urine

III. *Somatic Neurological Disturbances*
A. General signs
   1. Headache (its locus is not a reliable index of the site of underlying lesion)
   2. Blurred vision, photophobia
   3. Internal strabismus, diplopia, 6th nerve palsy
   4. Vertigo, in-co-ordination, tinnitus
   5. Epileptiform and decerebrate (tonic) seizures
B. Meningeal signs (often due to subarachnoid blood)
   1. Headache, photophobia
   2. Nausea, vomiting
   3 Fever (101°–103° F.)
   4. Stiff neck, extensor hypertonus
   5. Kernig and Brudzinski signs
   6. Moderate leukocytosis
C. Focal signs, expressed as dysfunctions of cranial nerves (8th, 7th, 6th, 1st, 3d and 2d nerves, in order of frequency involvement)
   1. Motor power and muscle status
   2. Reflexes, deep and superficial
   3. Co-ordination
      a) Equilibratory
      b) Nonequilibratory
   4. Sensation
      a) Superficial (pain, tactile, warm and cool) modalities
      b) Deep (position and vibratory) modalities
      c) Special (visual, auditory, gustatory, olfactory and equilibratory) modalities

The foregoing neurological signs may be thought of as subtended by *irritative* or *paralytic* lesions. The former (irritative) appear as exalted or perverted functions (e.g., as hypersensitivity, muscular twitchings and focal or generalized seizures) and commonly signify early or incomplete neural damage; the latter (paralytic) appear as depressed functions (e g., hyposensitivity, dyspraxia, dysarthria, paresis or paralysis) and commonly signify late or complete neural damage. Characteristically, there is much overlapping of irritative and paralytic

servative measures described below and proceeds to observe the patient's progress closely, re-examining him as often as changing conditions require and comparing the results of each examination with those previously recorded (Fig. 149). If it is apparent from the outset that a surgically amenable lesion exists, and the patient's general con-

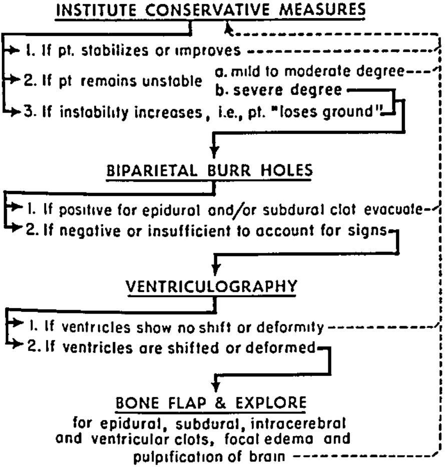

FIG 149.—Flow chart of management of the patient with acute head injury.

dition permits, operation is performed early, otherwise, conservative measures are employed until a more favorable condition is achieved. If, however, the patient appears at the outset to require no surgery, conservative measures are instituted and continued until maximal functional recovery is achieved. From this point on, only two circumstances prompt departure from the conservative program. (1) progressive "loss of ground" at any time and (2) failure after the first four to six days to "gain ground." If the patient exhibits either of the latter courses,

pressure; and fall in pulse rate. This is certainly the best known, and probably the most reliable, of the craniocerebral traumatic syndromes.

The second syndrome (increased intracranial pressure), like the first, has gained a seemingly secure place in the textbooks. It is that of brain compression due to increased intracranial pressure. It was asserted by Kocher (1899), Cushing (1903) and many subsequent investigators that from observations of rising blood pressure, falling pulse rate, slowing respirations and deepening stupor the clinician may reliably infer the existence, degree and direction of progress of increased intracranial tension. It was further claimed (and in some clinics still is) that these data furnish dependable criteria as to the therapy indicated, whether conservative or operative.

The unreliability of such syndromes, the reasons of their ineptitude and a citation of illustrative errors that have resulted from reposing false confidence in them have been amply demonstrated in previous clinical and experimental studies by Browder and Meyers (1936–42). Even greater uncertainty attaches to the more nebulous "syndromes" of acute subdural hemorrhage and traumatic subarachnoid hemorrhage.*

Recent writers have generally been impressed with the limitations of the syndromic concepts and have urged that "good clinical judgment," rather than adherence to rules-of-thumb, constitutes the preferred orientation in caring for patients with craniocerebral trauma.

The foregoing may be summarized as follows: Clinical signs and symptoms, even when they are consistent with a clinicopathologic syndrome, do not *make* the pathologic diagnosis; they merely increase the likelihood that the lesion associated with that syndrome will be found.

Management of the Head-Injured Patient

Fortunately, there is an effective way of circumventing the difficulties of envisioning the precise pathologic process(es) at work in a given patient This way is loosely referred to above as "good clinical judgment." We may now consider in what this judgment consists.

Briefly, the clinician confronted by a head-injured patient carries out a preliminary examination, administers first aid, institutes the con-

*The syndrome of chronic subdural hematoma is better defined than that of acute subdural hematoma, however, since the manifestations of chronic hematomas do not appear until a week or more after injury, their description does not properly belong in a discussion of acute head trauma.

SHOCK.—If shock is present, its management takes precedence over that of concomitant conditions. The standard measures for combating shock have been set forth above (Chapter 6). Morphine, often considered routine in the treatment of shock, is contraindicated in head injuries. Similarly contraindicated is the practice of lowering the head below heart level. Such posture encourages intracranial venous stasis and cerebral hypoxia.

Shock ascribable to head injury per se is not commonly encountered; for, if the initial shock produced by head trauma is not severe enough to kill quickly, recovery from the shock is rapid. Hence, by the time the patient reaches or is reached by medical help, he is usually out of shock or rallying rapidly from it. Among the approximately 6–8 per cent of the head-injured patients who present the picture of shock at the time of hospital admission or develop it shortly thereafter, the majority have serious extracranial injuries, such as abdominal or thoracic hemorrhage, fractures, etc.

TRANSPORTATION.—Once transportation has been decided upon, two practical points require consideration: (1) in being transferred the patient should be moved smoothly and firmly in "log-fashion." To so transfer the patient usually requires the co-ordination of three persons, acting on prearranged signals. (2) During the trip he should be kept in the Schafer posture.

POSITION.—Upon arrival at the hospital the patient should be put in a quiet room and placed well over on his side. The head of the bed should be raised about 20 degrees above the horizontal. The patient should be turned, in log-fashion, from side to side every two hours in order to prevent hypostatic pneumonitis and pressure sores.

PROTECTION.—Confused, refractory, hyperactive and partly stuporous patients may imperil themselves by climbing or falling out of bed; by removing dressings, oxygen catheters, nasogastric tubes, etc.; and by thrashing about in bed. Sedation in the usually recommended doses may not prove effective. Unless a capable nurse is continually in attendance, side boards and restraints will be required in such cases. The camisole is the most effective and innocuous form of physical restraint. Somewhat less satisfactory are leather cuffs or muslin bandage ties applied to the wrists and ankles. In using cuffs and ties, proper underpadding and secure anchorage to the side of the bed *without slack* are essential.

VITAL SIGNS.—Readings of the blood pressure, pulse rate, respirations and temperature should be recorded every fifteen minutes as long as significant variations are in evidence. When physiologic stabilization

surgical inquiry is indicated, i.e., trepanation, with or without ventriculography. Then, depending on the outcome of the inquiry, definitive surgery may or may not be indicated.

CONSERVATIVE MEASURES.—At the initial examination the physician endeavors to establish a "base line" consisting of a record of *vital signs* (color, pulse rate, respiration, blood pressure, temperature), *state of responsiveness* and *general neurological status*. All subsequent findings are referred to this and to one another. The preliminary examination is necessarily brief, consisting of a rapid appraisal of such data as can be obtained by inspection, palpation and manipulation. Pallor, rubor, cyanosis, sweating and palpable temperature of the skin can be noted within the first few moments; and the rate, regularity and force of the pulse and respiration can be estimated. A general idea of the existing state of consciousness can be reached by determining the promptness, vigor, co-ordination and adaptiveness of the patient's responses.

The patient should be inspected for evidences of active and arrested hemorrhage from the aural, buccal and nasal orifices. At the same time, tests for nuchal rigidity can be made. Lacerations of the scalp and general integument and gross evidences of fractures, dislocations and concealed injuries and hemorrhage should likewise be sought.

FIRST AID.—If active hemorrhage is present, it should be stopped. Neighboring parts should be shaved when necessary; simple wound treatment should be carried out; and a snug, dry gauze dressing applied. For the most part, the lesions described above under "Marks of Violence" (p. 656) can be disregarded during the first few hours.

The only other condition that demands immediate attention is impending asphyxia. This can usually be combated by arranging the patient's position so that the tongue, pharyngeal secretions, blood and vomitus gravitate away from the pharynx. The classical Schafer resuscitation posture * is satisfactory for this purpose.

The neural and humoral dysfunctions set into motion by partial asphyxia can lead rapidly to irreversible brain damage and death. Serious degrees of cerebral hypoxia may exist without cyanosis of the skin and mucus membranes. When cerebral hypoxia persists, its early systemic consequences (rise in blood pressure, increase of pulse pressure, fall in pulse rate and deepening of respiration) may be mistaken for those resulting from increase of intracranial pressure.

*In the Schafer position the patient is placed prone and the head turned to one side. The forehead rests on the forearm of the limb on the side opposite that toward which the face is turned.

livered, clinical experience demonstrates that, on the one hand, linear and stellate fractures may occur without neurological or psychologic dysfunction and, on the other hand, severe and fatal brain injuries may occur without accompanying skull fractures. The therapeutic regime adopted in a given case depends on what is conceived to be amiss with the brain, or with what may ultimately arise therefrom—e.g., infection, cicatrices, paralyses, convulsions, etc. This does not imply that x-ray examination need not be made. On the contrary, the demonstration of depressed and indriven bone fragments, foreign bodies, compound fractures of the sinuses and linear fractures across the course of the middle meningeal artery may prove of considerable value; and certainly, medicolegal considerations themselves make x-ray studies mandatory. *Nevertheless, it is rarely necessary to hurry the newly* admitted patient to the x-ray table, particularly if his general condition is considered precarious. In the majority of cases, radiologic examination may be postponed until the third or fourth day.

Laboratory Studies.—Soon after admission, a routine urinalysis and a complete blood count should be carried out. In the more severely injured cases, blood sugar and blood urea nitrogen determinations are desirable. Serological tests should be routinely carried out on the blood and spinal fluid, and, if indicated, the alcohol content of the blood and urine may be determined.

Temperature Control.—One of the conspicuous clinical features of the severely brain-injured patient is the development of an anhidrotic fever. This of itself may lead to death. With the sweating function deranged, *heat from the body must be dissipated* by means of respiration and the physical mechanisms of conduction, radiation and convection. At ordinary room temperatures and in hot weather, the latter are not sufficient. Every precaution must therefore be taken to keep hyperthermia under control. If successive readings of rectal temperature are near normal and stabilized, or falling toward normal, a temporizing policy may be adopted. If possible, the room temperature should be held at 55°–60° F. But if the rectal temperature exceeds 102.5° F., active measures must be employed to combat the fever. The patient should be stripped of bedclothes and an electric fan played continuously upon his exposed body. Sponging with a half-and-half solution of alcohol and water should also be instituted. These measures must be continued until the rectal temperature falls below 101.5° F. They are to be reinstituted whenever *it rises* above 102.5° F.

If, despite these measures, the febrile state continues to rise,

is established, the time interval between successive readings may be increased.

A slow pulse is not uncommon, even after mild head injuries; and it may persist for weeks. In itself, it does not constitute a bad omen. The same may be said of respiratory rates ranging as low as 12–14 per minute. However, blood pressures above 160/100 and less than 80/50, and—even more important—hourly fluctuations of blood pressure within these limits, constitute serious untoward signs. Similarly, instability of the pulse and respiratory cycles in which the rate and depth build up to a peak and then taper off every few minutes is often a forerunner of the Biot or Cheyne-Stokes patterns.

PULMONARY VENTILATION.—*If proper respiration is maintained, the* neurocardiovascular mechanism can be relied on to carry out its functions with almost machine-like efficiency. As soon, however, as hypoxia is permitted to develop, untoward circulatory changes develop which tend, in time, to break over into circulatory collapse. Where oxygenation is satisfactory, there is no necessity for administering pharmaceutic support to the cardiovascular system in the form of digitalis, caffein, epinephrine, nor-epinephrine, ephedrine, etc.

The on-the-side and Schafer postures, recommended above, facilitate respiratory exchange by allowing the tongue to gravitate forward. If mucus collects in the pharynx and larynx, it should be promptly aspirated. *Lax or edematous lips and nasal obstruction due to dried* blood in the nostrils may mechanically impair ventilation, resulting in a slow build-up of oxygen debt. In such cases, an oropharyngeal airway often allows prompt restoration. Vegetative instabilities and neurological dysfunctions often regress promptly upon proper establishment of the airway. Slight adjustments of the head on the pillow often make the difference between effortful and relaxed breathing.

If indicated, oxygen should be administered, preferably by the nasal catheter technic. In the case of infants and small children, an oxygen box is often a more convenient device. Tracheotomy should be promptly resorted to if the simpler measures for accomplishing *toilet of the pharynx and trachea prove ineffectual.*

X-RAY STUDIES.—If, during the first few days, the patient's condition remains satisfactory, the clinician need not be overly concerned regarding whether or not a skull fracture exists. The patient's condition depends much less on the skull injury than on concomitant cerebral damage. While it is, in general, true that the presence of a skull fracture is a rough quantitative index of the physical violence de-

vascular mechanisms; (*c*) they increase intracranial tension by 50–120 mm. H_2O, even in the normal individual; (*d*) they exert undesirable side effects, e.g., on the gastrointestinal and diuretic-antidiuretic functions; and (*e*) their sedative effects can be duplicated by less nocuous drugs.

LUMBAR PUNCTURE.—Lumbar puncture provides useful information as to (*a*) the presence or absence of blood in the spinal fluid, and, if present, approximately how much; and (*b*) the pressure of the cerebrospinal fluid.* Diagnostic puncture and manometry may be safely carried out on recently injured patients unless blood and/or spinal fluid is being discharged from the orifices. In the latter circumstance, the possibility exists of reversing pressure gradients between the intracranial contents and the extracranial atmospheric pressure, and thus of further exposing the injured portal to infection.

The withdrawal of a small amount of cerebrospinal fluid may give the clinician a useful anticipating mental "set" for the evaluation of subsequent developments. Thus, if the spinal fluid is water-clear and under normal pressure, it is extremely unlikely that cerebral laceration or contusion and subarachnoid, subdural, intracerebral or ventricular hemorrhage(s) are present. If, then, the patient should subsequently lose ground, it is highly suspected that he harbors an epidural clot, particularly if the spinal fluid remains clear but is now under increased pressure. If, on the other hand, the spinal fluid proves, on first encounter, to be bloody and under normal or near normal pressure, the inference is that cerebral contusion, laceration and/or subarachnoid hemorrhage(s) exist. If such a patient should subsequently lose a small amount of ground, the necessity for pressing inquiry concerning a surgically amenable lesion would be less urgent than in the first case.

Current opinion among neurosurgeons is that the severity of cerebral trauma can seldom be inferred from so simple a criterion as intracranial hypertension; still less, that measurements of intracranial pressure can be made a valid index as to the mode of therapy required.

The therapeutic value of lumbar puncture, as contrasted with the diagnostic, remains a matter of warm controversy. Some investigators contend that intracranial hypertension and cerebral edema can be successfully controlled, and blood eliminated from the cerebrospinal fluid, by repeated spinal taps. Others deny that such virtues attach to repeated taps, and warn of their potential dangers, contending

*The Queckenstedt test must not be done. See page 654.

colonic instillations of cool water are indicated. A necessary preliminary to instituting the latter is the simultaneous determination of rectal and axillary temperatures. This establishes an axillary equivalent of the rectal temperature. A colonic tube may then be inserted about 18 inches into the colon and 3–5 L. of cool tap water introduced. The water is allowed to remain there for about eight minutes; then it is siphoned off; and the measure is repeated over and over again. Meanwhile, large doses of aspirin may be administered. Should all these measures fail, resort must be made to ice packs. In principle, however, the clinician must act in accord with the conviction that in head injuries, as in heat stroke, hyperthermia must be effectively controlled; otherwise the fight will be lost on this ground alone.

Orificial Bleeding.—Bleeding and the escape of cerebrospinal fluid from the nose and/or ear need not excite undue concern. In most instances the discharge ceases spontaneously within a few hours or days. The involved orifice may be washed or swabbed out, as occasion demands. Packing and instillations and irrigations of medicinal agents are dangerous because they may establish a pyogenic meningitis. The patient's posture should be such as to favor gravitational drainage. If conscious and co-operative, the patient should be urged to suppress coughing and sneezing. Broad-spectrum antibiotic therapy should be instituted as a precaution against the development of infection.

Chemotherapy, Antibiotics and Serotherapy.—When infection is actually or potentially present, antibiotic and/or sulfonamide therapy should be administered. Whenever indicated, the patient should be adequately protected against tetanus and gas infection.

Sedation.—The stuporous and moderately restless patient requires no sedation. Severe restlessness, however, should be combated by sufficient doses of a drug to prevent the patient from injuring and exhausting himself. The following agents, used singly or in combination and administered by mouth or rectum, are useful: phenobarbital, 60–200 mg.; bromides, 1.5 Gm.; chloral hydrate, 1.5 Gm.; and paraldehyde 4–12 ml. every four hours as needed. Very disturbed patients require barbiturates intramuscularly or intravenously.

It is useful to bear in mind that a distended bladder is capable of provoking restlessness and that catheterization offers prompt relief.

Narcotics should be circumspectly avoided for the following reasons: (*a*) their miotic effect precludes the development of one of the most valuable of "localizing" signs, viz., a disparity in the size of the pupils; (*b*) they depress the activity of the respiratory and cardio-

juice. In addition, a simple humidifier may be employed in the form of a water-moistened double layer of gauze laid across the external air passages.

ELIMINATION.—Urinary incontinence is a frequent complication in stuporous and disturbed patients. If bedsores are to be averted, it is essential that linen be changed promptly whenever soiling occurs. An indwelling Foley catheter may be passed into the bladder and connected directly to a drainage bottle or to an intermittent drainage apparatus. Urinary retention, which is much less frequently encountered, should be managed by the same devices.

During the first two to three days following head injury, little attention need be given the bowels. Incontinence of stool does occur, but not nearly so frequently as urinary incontinence. On the other hand, fecal impaction is prone to develop in unconscious and bedridden patients. It should be suspected whenever abdominal distention or constipation appears.

CONVULSIVE SEIZURES.—Although dramatic, convulsions do not in themselves necessarily constitute a serious prognostic sign. This statement holds particularly true in regard to children and the isolated, occasional seizures in adults.

Once a convulsion has started, it cannot be readily aborted—nor need it be. The only procedures immediately necessary are those concerned with protecting the patient from asphyxia and self-injury. There is no necessity for prying the jaws apart in order to gag a mouth that is already clenched in spasm. Injury to the lips, tongue and teeth may result. However, repeated convulsive attacks (status epilepticus) lead rapidly to exhaustion and demand anticonvulsant measures in the form of bromides or dilantin sodium by mouth or barbiturates by vein or muscle If the drugs mentioned prove insufficient, ether inhalation or rectally administered Avertin® (90 mg./kg. of body weight) should be employed

It is important that the observer record accurately the march of motor events that characterizes the onset of a convulsive attack. Such data may furnish valuable localizing clues, which in turn may prove of value in surgical therapy

BEDSORES—It is essential to exercise unceasing vigil against bedsores and hypostatic pulmonary congestion. To these ends, the patient should be turned from side to side every two hours. Susceptible pressure areas should be gently massaged, and bed linen changed as soon as soiling occurs. Sheets should be kept smooth at all times.

that reduction of intracranial pressure may permit recently occluded vascular channels to reopen, result in medullary and hippocampal herniations and provoke an undesirable overproduction of cerebrospinal fluid. They assert that "nondisplaceable" elements, like cerebral edema and collections of blood, soon take up the space previously occupied by the displaceable cerebrospinal fluid and that this effectually reduces the patient's margin of safety.

DEHYDRATION.—Following Weed's demonstration (1921) that hypertonic saline administered intravenously to normal dogs is capable of reducing intracranial tension, it became standard clinical practice to employ parenteral dehydrating agents in the treatment of head injuries. In addition, the daily fluid intake was commonly restricted to 600–1,000 ml. per day, and magnesium sulphate was administered by mouth. Through the years, hypertonic saline has been successively replaced by glucose, sucrose, sorbitol and concentrated serum albumin. About fifteen years ago, serious warnings of the dangers inherent in dehydration therapy began to appear. Skeptics asserted that intracranial hypertension of magnitude sufficient to constitute a clinically significant factor is the exception rather than the rule; that the effect of hypertonic solutions is not only unpredictable but may prove detrimental; that serious renal damage may be produced; that water depletion in the presence of hyperthermic toxic states carries serious threats, and that a fatal "reverse osmosis" * may occur.

In brief, the principle of vigorous dehydration appears to possess little merit when employed in the head-injured patient and is gradually being abandoned. The daily fluid intake should generally exceed 1,800–2,000 ml.

NUTRITION.—Unconscious and restless patients should not be carried for more than twenty-four to forty-eight hours on parenterally administered fluids alone. If at the end of such period they are still unable to take nourishment by mouth, a nasogastric polyethylene tube should be passed and stomach feedings instituted.

MOUTH HYGIENE.—Unconscious patients breathing for hours on end through the open mouth are prone to develop parotitis. The mouth should be cleansed of dried saliva every three to four hours. A good solution for this purpose consists of equal parts of glycerine and lemon

*When the finer vascular radicals of the brain have been interrupted by laceration, contusion and multiple thromboses, the passage of hypertonic solutions is blocked, and the hypertonic agent can spill out of the injured vessels, infiltrate the interstitial tissues, imbibe watery fluids and further intensify the pre-existing edema.

2. Simple depressed fractures of the vault—such that the outer table of the depressed portion is at, or deeper than, the inner table of the adjacent vault
3. Epidural hemorrhage
4. Subdural hemorrhage or hemohygroma
5. Massed intracerebral and intraventricular clots
6. Lobar pulpification of brain substances

The first two of these, which are, in most instances, apparent or strongly suspected from external signs alone, rarely present diagnostic difficulties. They are readily confirmable by x-ray. Of the last four, epidural hemorrhage is the most urgent. Lack of treatment for an hour or two can prove fatal. The therapeutic issues between conservative and definitive surgical measures can be met with fair certainty by making a burr opening over the parietal bosses. Should an epidural or subdural clot be encountered, it should be evacuated; if no lesion is disclosed, the surgeon must pursue matters further, injecting air into the ventricles and taking x-rays (ventriculography). If the ventricular shadows appear normal, he may conclude that further operative measures are unnecessary and reinstitute conservative measures. But if there is a ventricular shift or distortion, he must reflect a bone flap and search for an epidural, subdural, intracerebral and/or intraventricular clot and gross pulpification of brain tissue. If edema only is encountered, the bone flap may be sacrificed or loosely replaced. However, nonspecific "decompressive" procedures of this type can rarely be relied on to serve a useful purpose, indeed, they may make matters worse, inviting further edema by occluding veins at the bony margins of the decompression.

After the patient has been brought through the acute phase of his injury, a number of residual conditions may require attention. Physical therapy and psychosociologic rehabilitation will be required for almost all patients with severe head injuries. This job calls for an integrated paramedical team. Post-traumatic epileptiform seizures may require sustained medical treatment over the course of years. In the surgical realm, treatment may have to be directed at chronic subdural hematomas, persistent cerebrospinal fluid rhinorrhea and otorrhea, arteriovenous fistulas with pulsating exophthalmos, convulsive states and defects in the vault of the skull. But, for the most part, all these are matters of election rather than urgency; and since they constitute sequelae and complications of a chronic character, they fall outside the scope of this discussion.

LOSS OF GROUND; FAILURE TO GAIN.—When the experienced physician concludes that the patient is holding, gaining or losing ground, he makes more or less deliberate use of certain categories of observation, viz., the *vital signs, the patient's responses* (state of consciousness) and the *neurological findings.* If repeated examinations indicate that the patient is gradually approaching a normal status in all three categories, conservative measures alone are in order. But if the patient fails to make perceptible gains after the fifth day, or if he exhibits *at any time* a progressive decay of responses to stimuli and instability of vital signs, surgical inquiry becomes mandatory. The decision to seek surgical help is reinforced whenever neurological deficits previously in evidence become more apparent and/or new deficits appear. The point to be stressed is that the physician can decide whether the patient is gaining, holding or losing ground *without* making a definitive pathologic diagnosis.

Instability of the vital signs is a more sensitive indicator of ensuing trouble than the patterns of the signs and symptoms of the classic clinicopathologic syndromes. As an isolated finding, a bradycardia of 50 to 60 beats per minute, even if persistent for days or weeks following head injury, need arouse no anxiety; nor should a tachycardia of 110 to 120 beats per minute, particularly if it occurs in children and young adults. Broadly speaking, relative stability of pulse rate, blood pressure, temperature and respiration, whether above or below normal, justifies temporization. On the other hand, wide fluctuations of the pulse, blood pressure and respiration indicate the need for surgical inquiry.

Neurological disabilities in the form of progressive pupillary disparities, nystagmus, conjugate movements, aphasia, paralysis, spasticity, twitchings and convulsive phenomena command particular attention. If the vital signs and the state of responsiveness remain satisfactory during the first few days, the advent of neurological disabilities needs not, per se, force surgical inquiry. But when such disabilities coexist with instable vital signs and deepening stupor, surgical inquiry becomes mandatory (Fig. 149).

SURGICAL INQUIRY FOR SURGICALLY AMENABLE LESIONS.—The lesions that demand surgical attention during the early days following head trauma are:

1. Compound fracture(s) of the vault—with or without depression, indriven bony fragments, foreign bodies, devitalized and/or extruded brain tissue

2. Simple depressed fractures of the vault—such that the outer table of the depressed portion is at, or deeper than, the inner table of the adjacent vault
3. Epidural hemorrhage
4. Subdural hemorrhage or hemohygroma
5. Massed intracerebral and intraventricular clots
6. Lobar pulpification of brain substances

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In the late management of seriously injured patients, the general practitioner and surgeon will usually find it expedient to seek the help of neurologic specialists.

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CHAPTER 31

Chest Injuries

PULMONARY FUNCTION consists of two phases: ventilation and respiration. Ventilation is the movement of gases to and from the lung as a result of the alternate increase and decrease in the capacity of the thorax. Inspiration, or movement of gases into the lung, is an active process resulting from contraction of the muscles of respiration, while expiration, or movement of gases out of the lung, is a passive process related to the elasticity of the lung and chest wall. The pulmonary phase of respiration (or external respiration) has to do with the absorption of oxygen and the elimination of carbon dioxide between the pulmonary alveoli and the blood in the pulmonary capillaries. The ultimate objective of this interchange of gases is to provide oxygen and remove carbon dioxide according to the metabolic needs of the tissues. All influences which interfere with operation of this delicately integrated system of gaseous transport and exchange *result in impaired cellular function.*

Normally, there is a potential space between the visceral and parietal pleurae. The negative intrapleural pressure, which at rest measures about −7 and −2 cm. of water on inspiration and expiration, respectively, maintains the pleural surfaces in contact and the lung in expansion. Alterations in intrapleural and intrathoracic pressure also play an important part in blood flow through the veins to the heart.

When air or fluid is introduced into the pleural space, the lung collapses. The pressure differential between the two pleural cavities *causes a shift of the mediastinum and a reduction in the ventilatory* capacity of the contralateral lung. Under these circumstances, respirations are exaggerated in rate and depth, according to the extent of the collapse and displacement. In spite of this, however, ventilation may be deficient and hypoxia develops. The combination of mediastinal

shift and decreased intrapleural pressure also causes interference with venous flow and cardiac filling.

Pneumothorax may occur spontaneously or from trauma (Fig. 150). *Closed pneumothorax* results from rupture of the lung or large

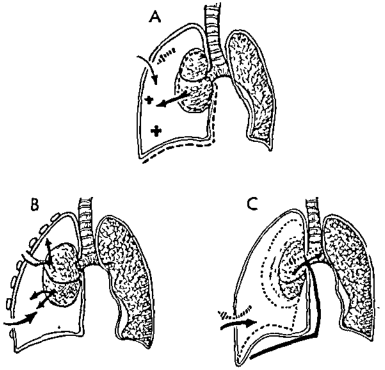


Fig. 150.—Varieties of pneumothorax. *A*, open pneumothorax produced by air entering the pleural space from the outside; closed pneumothorax produced by air entering the pleural space from the lung. Closed pneumothorax is likely to be complicated by "tension pneumothorax" because the opening in the lung may act as a one-way valve. Combined open and closed pneumothorax is often the result of penetrating wounds (e g , bullet, stab, etc.). *B*, closed pneumothorax due to puncture of the lung by a fractured rib. Combined pneumothorax produced by a penetrating wound. *C*, large, open pneumothorax ("sucking wound of the chest"). Air enters and leaves the pleural cavity with respiration. The mediastinum shifts back and forth, and air passes to and fro from the intact to the partly collapsed lung (*pendelluft* phenomenon). There is little exchange through the normal airway. Asphyxia results.

air passages and air which enters the pleural space. In *open pneumothorax,* an opening in the chest wall allows air to enter the pleural cavity from the outside. Combined chest wall and lung injuries may permit air to enter the pleural space from both sources. When air enters the pleural cavity with inspiration and is unable to leave (one-way valve action), intrapleural tension mounts and a dangerous con-

dition known as *tension* (or *pressure*) *pneumothorax*, is produced.

When open pneumothorax results from a wound large enough to allow air to enter and leave the pleural cavity with each inspiratory and expiratory effort, the condition is known as a *sucking wound of the chest*. If the opening approaches or exceeds the size of the normal airway (trachea), the resulting disturbance in ventilation will rapidly lead to death by asphyxia unless the opening in the chest is closed. In this condition, movement of air takes place between the opened pleural space and the exterior, as well as between the collapsed and the normal lung (*pendelluft* phenomenon) rather than between the atmosphere and the lungs. The opening in the chest wall must be closed before adequate ventilation and respiration can be restored.

A somewhat comparable situation results from an extensive thoracic injury with fractured ribs and unstable chest wall ("flail chest"). Here an interchange of unoxygenated air occurs between the lung of the injured side and the lung of the intact side, because on inspiration the flail side is drawn in or retracts rather than expands (paradoxical respiration). Furthermore, because movement of the chest wall is painful and the mediastinum is unstable, both ventilation and respiration are greatly impaired.

When large amounts of blood accumulate in the pleural space (*hemothorax*), cardiorespiratory disturbances resembling those produced by air result. There is a reduction in the aerating surface of the lung, compression of the opposite lung from mediastinal shift, and reduced cardiac filling. Under these conditions there is a marked predisposition to shock, which is enhanced by the loss of blood from the vascular compartment.

When air extravasates into tissue planes from lacerations of air-containing organs, *emphysema* is produced. Such collections of air may appear subcutaneously (*subcutaneous emphysema*), where they may remain localized or may become diffused. Generally, they cause no difficulty and the air is gradually absorbed. When air collects in the mediastinum (*mediastinal emphysema*), compression of the great blood vessels and tracheobronchial structures may result.

Traumatic wet lung is the result of severe chest injury with pulmonary contusion. There is intrapulmonary hemorrhage and edema, stasis with retention of secretions, and infection. The patient ultimately may "drown in his own secretions." Atelectasis and infection develop, and the patient succumbs to bronchopneumonia unless vigorous measures are initiated to prevent this sequence of events.

Traumatic asphyxia is produced by sudden and violent compression of the chest with marked back pressure into the systemic veins, particularly those of the head and neck. The physiologic alterations are those which might be imagined to result from the Valsalva maneuver extended far beyond physiologic limits. There is cyanosis, hemorrhages of the head and neck, brain damage and sometimes death.

Cardiac tamponade results when blood escapes from the heart more rapidly than from the pericardial sac. As intrapericardial pressure increases, the flow of blood to the heart is impeded and cardiac output falls. The immediate therapeutic problem concerns relieving the pressure by aspiration or operation and restoring normal cardiac function.

Examination of Chest

The extent of injury may be difficult to assess on the initial survey. Therefore, frequent and repeated examinations are usually necessary. In some patients, the damage may be predicted on the basis of the traumatic agency and its path (e.g., gunshot wound); in others, the extent of damage is more obscure. Compound injuries are common. The usual studies include a general history and physical, x-ray, electrocardiogram, blood and urine examinations.

Inspection of the chest may reveal abrasions, contusions, perforations, lacerations or loss of substance of the chest wall. Deformity of the rib cage and sternum and impaired excursion on inspiration are often apparent. In minor injuries, tenderness, deformity and bony crepitation may be localized discretely over one or several ribs. In the flail (stove-in) chest, multiple fractures of ribs—sometimes those of the entire hemithorax—may be noted. In this condition there may be "paradoxical respiration"; that is, the movement of the thoracic cage on the injured side is opposite to that on the uninjured side, or inward on inspiration and outward on expiration.

Percussion over the injured chest may indicate the presence of pneumothorax (hyper-resonance) or hemothorax (dulness). If the mediastinum and trachea have shifted toward the intact side, increased intrathoracic pressure on the injured side (e g., fluid, air, or both) should be suspected. When the mediastinum has shifted toward the involved side, decreased intrathoracic pressure due to atelectasis or massive collapse should be suspected

The breath sounds may be tubular in the presence of small amounts of intrapleural fluid or air, or absent when large amounts

occupy the pleural space. Fine râles suggest pulmonary congestion; coarse râles, excessive tracheobronchial secretions with stasis. If there is a precordial "crunch" or "click" which is synchronous with the heartbeat, mediastinal emphysema is probable. When bowel sounds are heard in the chest, herniation of abdominal viscera through a ruptured diaphragm must be suspected.

Cardiac injury may be indicated by the irregularities in the pulse, alterations in the heart sounds or electrocardiographic changes. Injury to the heart may result from contusions or closed injuries (e.g., steering-wheel injury) and frequently are unrecognized.

X-RAY EXAMINATION OF CHEST

In an emergency, a working diagnosis based on the history and physical examination may be adequate, but x-ray films are essential before definitive treatment. The films should be made with the patient in the sitting position if possible, and both anteroposterior (or posteroanterior) and lateral exposures are desirable. Repeated films are necessary to follow the progress and to guide therapy after injuries to the chest.

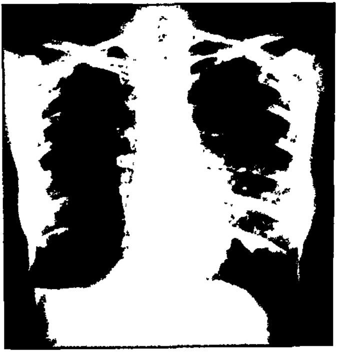
Fractures of the ribs are usually evident but may be overlooked unless searched for in the injured area. Costal cartilage fractures cannot be seen on the x-ray film. The lung fields, the mediastinum and the diaphragm and ribs should be carefully inspected. Pneumothorax is indicated by the absence of lung markings and the separation of the visceral pleura from the parietal pleura by air. Increased density of the lung fields may be produced by fluid in the pleural cavity. When fluid but no air exists in the pleural cavity, it is difficult to detect the fluid on the x-ray in amounts less than 300 ml. When both fluid and air are present, a "fluid level" is seen when the patient is x-rayed in the upright position (Fig. 151). Under these conditions, small amounts of intrapleural fluid can be recognized. Enlargement of the cardiac silhouette suggests cardiac injury, hemopericardium and, possibly, cardiac tamponade.

GENERAL PRINCIPLES IN TREATMENT OF CHEST INJURIES

The status of cardiorespiratory function is the primary concern after all chest injuries. There are simple and readily available methods for supporting and sustaining vital functions, provided the need is

recognized. It is important, therefore, that the physician possess a basic understanding of the common acute disturbances of the chest and the measures which are effective in combating them.

Shock from chest injuries is more often the result of deranged cardiorespiratory function than of blood loss. Painful respiratory movements lead to poor ventilation and hypoxia. When extensive damage to



The patient made an uneventful recovery

the thoracic cage exists, these factors may produce or aggravate shock. Of course, acute hypovolemia from blood loss should also be kept in mind. Treatment for shock must be instituted promptly, and every effort made to maintain vital functions.

Pain may be controlled by support of the chest wall and procaine intercostal nerve block. Opiates must be used sparingly, if at all, because they depress ventilation and the cough reflex. They are best given intravenously in small doses, in order to secure the desired effect immediately. Blood volume must be restored by whole-blood trans-

fusion, with due consideration to the dangers of overloading the circulation if ventilation is impaired.

The patient should be examined completely for multiple or combined injuries. Thoracic injuries are frequently associated with other regional soft tissue, skeletal or visceral injuries.

The general principles in the treatment of chest injuries are:

1. Combat shock; correct disturbed cardiorespiratory function; relieve the pain; replace blood loss. Avoid depressant drugs.
2. Enhance the efficiency of aeration. Maintain a good airway; administer oxygen if necessary; place the patient in the position most conducive to effective ventilation.
3. Correct the cardiorespiratory disturbance. Cover the sucking wound; decompress tension pneumothorax; relieve mediastinal pressure or shift; support the painful and unstable chest wall; relieve pain.
4. Do not move the patient unnecessarily.
5. Obtain chest x-ray films for diagnosis and as a guide to treatment.
6. Treat wounds and associated injuries.
7. Avoid pulmonary edema. Administer parenteral fluids cautiously.
8. Prevent infection by proper wound treatment and use of antibiotics and indicated prophylactic antisera and/or toxoid.

Technics in Treatment of Chest Injuries

Intercostal Nerve Block.—From 5 to 10 ml. of 1 per cent procaine may be injected into the region of the intercostal nerves, including two nerves above and two nerves below the area of injury. The injection should be made just lateral to the erector spinae muscle. Care must be taken to avoid injuring the intercostal vessels or entering the pleurae, producing a pneumothorax. The block may be repeated if necessary.

Adhesive Strapping of the Chest.—This is a simple and effective method for supporting fractured ribs and a damaged chest wall. It may not be applicable in extensive injury. The skin of the hemithorax should be shaved and protected by applying a coating of tincture of benzoin. Adhesive tape (preferably the elastic type), 4 inches in width, is then applied with the chest in expiration, allowing the tape to extend at least 2 inches beyond the midline both in front and in

back. The tape may be left in place for five to seven days, and may be reapplied if necessary.

THORACENTESIS —Thoracentesis (aspiration of air or fluid from the pleural space) may not be necessary to remove small amounts of air or fluid if there is no ventilatory difficulty. *Aspirate under aseptic precautions only.* Air may be aspirated from the pleural cavity by *introducing a needle high in the axilla, or in the second interspace* anteriorly, after local infiltration of the skin with procaine. In the presence of continued leakage or tension pneumothorax, an intercostal catheter should be introduced and connected to a water-seal system. The glass tube of the system should extend at least 5 cm. below the level of water in a bottle which is placed on the floor beside the patient's bed. This apparatus acts as a valve which permits air to leave the system but prevents air from entering it. Because of the danger of water entering the pleural space, the bottle should never be elevated above the floor unless the tubing has been clamped.

Fluid in the chest should be removed by aspiration, with the patient in the sitting position. For this procedure the skin is anesthetized and the needle is introduced into the dependent part of the fluid collection, usually at the level of the eighth interspace in the scapular line. If the patient complains of tightness in the chest, faintness or vertigo, or has uncontrollable cough, pallor or rapid pulse, the procedure must be discontinued. If there is no undue reaction, as much as 1,000–1,500 ml. of fluid may be removed. The fluid should not be replaced by air. Repeated aspirations may be required, or tube drainage with water-seal bottle drainage may be necessary. Obtain bacteriologic studies on the aspirated fluid if indicated.

TRACHEOBRONCHIAL ASPIRATION.—This is a simple and effective measure for clearing the upper air passages of secretions and exudate. It is performed as follows: The patient is placed in the semisitting position. A new, preferably "whistle tip," No. 16 or No. 18 F. catheter, connected by tubing to a suction machine, is used. The catheter is introduced (without suction) through the nose as the tongue is pulled forward. When the patient inspires, the catheter is rapidly advanced, the tongue is released and suction is started. The catheter is moved up and down for a few seconds, and then withdrawn a short distance and reinserted. If gagging occurs, the tube is probably in the esophagus; if there is stridor and coughing, the tube is in the air passages. The procedure may be repeated at frequent intervals.

If a catheter aspiration proves ineffective, bronchoscopy or tracheot-

omy with frequent aspirations may be needed to maintain the airway.

TRACHEOTOMY.—Chest injuries may be associated with neck, jaw or head *injuries which contribute to respiratory* obstruction and retention of secretions. It is imperative that the airway be kept open and clean if the patient is to survive. If this cannot be accomplished by other means, *tracheotomy must be done without delay.* To procrastinate is to risk the patient's life.

Emergency tracheotomy may be necessary. For this procedure the head is placed over the side of the bed with the neck in extension. A longitudinal incision is made in the midline of the neck over the trachea. The *fascia is separated in the midline, and an opening is* made into two tracheal rings *at least two rings below the cricoid cartilage.* A tracheal cannula or an improvised device is inserted to maintain the opening in the trachea. Catheter aspiration should be carried out at frequent intervals. (For the details of post-tracheotomy care, which are very important, see other texts.) In elective tracheotomy, a transverse skin incision and other refinements in the performance of this operation are preferred.

TRANSTHORACIC OPERATION.—*This operation may be indicated in* open chest wall wounds, continued intrathoracic bleeding, combined thoracoabdominal injuries, esophageal injuries, progressive and rapid accumulation of air in the thorax and certain heart injuries.

COMMON THORACIC INJURIES AND TREATMENT

SIMPLE FRACTURES OF THE RIBS.—These are usually produced by direct trauma. The patient complains of local pain, *aggravated by* cough or deep breathing. There is tenderness, a palpable deformity and often crepitation at the fracture site. The diagnosis is confirmed by *x-ray examination. Adhesive strapping,* intercostal nerve blocks, analgesics and rest are effective therapeutic measures.

OPEN PNEUMOTHORAX.—In open pneumothorax (sucking wound), the larger the opening in the chest wall the smaller the amount of air that enters the lungs through the trachea. The lung on the injured side collapses, and the mediastinum shifts toward the good side and tends to move back and forth with respiration (mediastinal flutter). Air also passes back and forth from one main bronchus to the other with each respiratory cycle (*pendelluft* phenomenon). *Ventilation is rendered* ineffective, and asphyxia ensues.

Immediate closure of the chest wound with an occlusive dressing

is imperative. When positive-pressure anesthesia is available, definitive treatment of the chest wound and underlying damaged lung and pleurae may be instituted.

Tension Pneumothorax.—When there is a laceration of the lung which allows air to enter but not to leave the pleural space, tension (or pressure) pneumothorax develops. This may follow trauma or spontaneous rupture of an emphysematous bleb of the lung. Progressive collapse of the involved lung, mediastinal shift and decrease in capacity and function of the opposite lung follow. Cardiac filling and output are impaired. Anoxia, with rapidly progressive deterioration of the patient, ensues.

The side of the collapsed lung is hyper-resonant. Breath sounds are absent. The trachea and mediastinal structures are shifted toward the opposite side. There is marked dyspnea with pallor or cyanosis. Expiratory efforts are shallow, and inspiratory efforts difficult.

The condition may be recognized from the clinical history and physical findings. X-ray findings are confirmatory.

Immediate decompression may be effected by the introduction of a large bore needle into the second or third interspace anteriorly, followed, if necessary, by a catheter (Nos. 20–22 F.) introduced through a trocar and connected to a water-seal apparatus.

Hemothorax.—Some blood in the pleural space is to be expected in nearly all chest injuries. Large collections which reduce the pulmonary reserve and deplete the blood volume lead to rapid deterioration of the patient's condition. The bleeding may come from vessels of the chest wall or the thoracic organs. Rapid hemorrhage can occur from the intercostal or internal mammary arteries. Less active bleeding, which may stop spontaneously, usually results from injuries to veins, small arteries or the lung. Hemothorax can be produced by both closed or open injuries. It should be remembered that the intercostal vessels may be torn by sharp ends of fractured ribs.

If the pleural space promptly refills with blood after aspiration, direct ligation of the bleeding vessel at thoracotomy may be required. Intermittent aspiration of blood is usually adequate in lesser degrees of hemothorax. Aspiration should be continued so long as about 100 ml. or more of fluid is obtained.

Blood in the chest usually remains fluid as a result of defibrination by the action of the heart and lungs. The fibrin is deposited on the pleural surfaces, where it eventually forms a thick fibrin covering, or

"peel." The peel gradually becomes organized fibrous tissue which incases the lung, prevents its expansion and renders it completely functionless. The main objectives of treatment in hemothorax are to remove the blood from the pleural cavity and to expand the lung as early and completely as possible. If this is not accomplished, decortication and expansion of the lung by operation may be necessary. Operation is usually not done sooner than three to six weeks after the development of hemothorax, or until a line of cleavage exists between the peel and the pleura; but it should be done before fibrous tissue organization has occurred.

If blood in the pleural cavity becomes infected, *empyema* (thoracic) develops. The exudate becomes purulent, and the patient exhibits systemic signs of infection. Bacteriologic studies will indicate the offending organism (or organisms) and the specific antibiotic therapy most likely to be effective. Intercostal tube drainage, or rib resection with closed (or open) drainage of the pleural space, may be required before the infection is eliminated.

FLAIL ("STOVE-IN") CHEST.—Multiple fractures of the ribs and/or sternum lead to loss of chest wall rigidity. Paradoxical movement of the chest wall, mediastinal flutter and the *pendelluft* phenomenon develop. Shallow grunting respirations with the inability to evacuate lung secretions and exudates by coughing are constant findings. Wet lung and other complications are to be anticipated unless the chest wall is stabilized, the pain is relieved and the tracheobronchial tree is cleared.

Intercostal blocks, stabilization of the chest wall, enforced coughing while the chest is supported, and tracheobronchial aspiration are helpful. Adhesive strapping is most useful if only one side is involved. In bilateral chest injuries, elevation and fixation of the sternum by traction, applied through a towel clip fastened to the sternum, may be advisable.

TRAUMATIC WET LUNG.—This condition probably develops to some degree in all serious chest injuries. It is characterized by a wet, ineffective cough, with rattles and wheezes often heard at a distance from the patient. There may be restlessness and cyanosis. Both fine and coarse râles are noted on auscultation. The x-ray findings are sometimes suggestive of bronchopneumonia or atelectasis, but they may be negative. The immediate problems and the therapy are: to improve tracheobronchial drainage—by aspiration, bronchoscopy, or both; to

relieve the painful respirations and cough—by intercostal block rather than with morphine; to relieve anoxia—with oxygen; and to prevent infection—by administration of antibiotics.

Blast Injury.—Blast injury is not commonly encountered in civilian life. It results when explosive forces produce disruption of the lung parenchyma with rupture of the pulmonary vessels and alveoli, intrapulmonary hemorrhages, edema, emphysema and loss of normal pulmonary elastic recoil.

There may be little external evidence of a chest injury; but dyspnea, cough, hemoptysis (bloody froth) and signs of diffuse pulmonary edema and pneumonitis may appear. The treatment is conservative: absolute rest, antibiotics and treatment for shock. Secretions should be removed by aspiration. Pulmonary edema may require positive-pressure oxygen inhalation and hyperosmotic solutions intravenously.

Mediastinal Emphysema.—In this condition, there is an accumulation of air in the mediastinum as a result of traumatic, operative or spontaneous perforations of air-containing organs, such as the trachea or bronchi, lung or esophagus. The extravasated air extends about the hilus of the lung, the great vessels of the mediastinum and subcutaneously into the head and neck. The pressure in the mediastinum may occasionally be sufficient to interfere with cardiopulmonary function.

In severe mediastinal emphysema, there are several important findings: The veins of the head and neck are distended. Cyanosis, anxiety and dyspnea are common. Crepitation at the base of the neck may be detected. Characteristically, there is a precordial crunch or click which is synchronous with the heartbeat. X-ray examination reveals air in the mediastinum and usually an associated pneumothorax. The tension may be relieved by making an opening into the mediastinum through a low "collar" incision, similar to that used in thyroidectomy. If pneumothorax is present, it must also be treated. If there is an esophageal wound, it must be treated surgically.

Subcutaneous Emphysema.—Air may also extravasate subcutaneously from mediastinal emphysema or from tension pneumothorax through a rib fracture site in the chest wall when the parietal pleurae is torn. The extravasation may be localized or widespread. Although the appearance of the patient is often frightening, this condition in itself is not serious and requires no active intervention. Treatment should be directed toward the primary underlying condition.

Acute Cardiac Tamponade.—Stab and bullet wounds may cause hemorrhage from the heart or coronary vessels into the pericardial sac. Pericardial tension increases from relatively small amounts of blood (200 or 300 ml.). First the superior and inferior vena cava, then the auricles, and finally the ventricles are compressed. The triad of acute cardiac compression (Beck) consists of (1) a quiet heart; (2) rising venous pressure and (3) falling arterial pressure. A shocklike picture develops, and the condition is fatal unless promptly relieved. The pericardial sac may be aspirated by introducing a long needle (Nos. 15–18) through the left costoxiphoid angle inward and upward (about 45 degrees) for a distance of about 4–5 cm. If symptoms and signs of tamponade are relieved, this may be enough; but if they recur, repeated aspirations or open operation for control of hemorrhage may be required.

Cardiac Contusion.—It is probable that contusion of the heart occurs more frequently than realized. Direct trauma to the chest wall may cause slight or severe cardiac damage. Contusion is suggested by irregularities of the pulse, alterations in the heart sounds and electrocardiographic changes. In many respects the condition resembles coronary occlusion, and the treatment is quite similar.

Thoracoabdominal Injury.—The mortality in combined injuries is greater than from wounds of either the chest or abdomen. When the probability of abdominal injury exists but the issue is not clear, exploration may be advisable. It should be remembered that pure thoracic injury may also cause severe abdominal signs. Abdominal spasm due to thoracic trauma is often localized to one side. Intercostal nerve blocks may aid in determining the relative importance of chest pain and abdominal signs. When cardiorespiratory function is seriously disturbed, it should, if possible, be improved before exploration is undertaken.

The patient with a chest injury can be said to be progressing satisfactorily when (1) bleeding has been controlled; (2) the patient is totally resuscitated; (3) the tracheobronchial tree has been satisfactorily cleared, (4) the chest wall is stable and painless to the extent that cough and aeration are effective; (5) no signs of circulatory disturbances are present; (6) all wounds are properly débrided and dressed; (7) the pleural space is free, or is being freed, of blood and/or air; and (8) the lung is expanded or expanding.

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